A2623-1

American Journal of Public Health

May 1991, Vol. 81, No. 5 Established 1911

Editorial

561 Serosurveillance of Human Immunodeficiency Virus Infection *T. J. Dondero, Jr., and J. W. Curran*

Commentary

563 Cuba's Response to the HIV Epidemic *E. J. Pérez-Stable*

Featuring AIDS/HIV

568 Effects of an Intervention Program on AIDS-Related Drug and Needle Behavior among Intravenous Drug Users *R. C. Stephens, T. E. Feucht, and S. W. Roman*

572 Adult Survivors of Childhood Sexual Abuse and Subsequent Risk of HIV Infection *S. Zierler, L. Feingold, D. Laufer, P. Velentgas, I. Kantrowitz-Gordon, and K. Mayer*

617 Evaluating Needle Exchange: Do Distributed Needles Come Back? *J. Guydish, G. Clark, D. Garcia, M. Downing, P. Case, and J. L. Sorensen*

619 Targeted HIV Screening at a Los Angeles Prenatal/Family Planning Health Center *L. J. Fehrs, D. Hill, P. R. Kerndt, T. P. Rose, and C. Henneman*

623 HIV-1 Prevalence in Selected Tijuana Sub-populations *F. Güereña-Burgueño, A. S. Benenson, and J. Sepúlveda-Amor*

625 HIV Infection in an Ethiopian Prison *Y. Kebede, J. Pickering, J. C. McDonald, K. Wotton, and D. Zewde*

628 Comparison of AIDS Knowledge, Attitudes, and Behaviors among Incarcerated Adolescents and a Public School Sample in San Francisco *R. J. DiClemente, M. M. Lanier, P. F. Horan, and M. Lodico*

631 The Donation and Sale of Blood by Intravenous Drug Users *D. D. Chitwood, J. B. Page, M. Comerford, J. A. Inciardi, C. B. McCoy, E. Trapido, and S. Lai*

650 Public and the Law: The Effect of Mandatory Premarital HIV Testing on Marriage: The Case of Illinois *J. McKillip*

654 Notes from the Field: Community Leaders Help Increase Response Rates in AIDS Survey *J. M. Longoria, N. H. Turner, and B. U. Philips*

(continued page 547)

APHA
American Public Health Association

Featuring AIDS/HIV

Contents

American Journal of Public Health

"Information for Authors" appears on page 134 in the January 1991 issue of the Journal and may also be obtained from the Journal Editorial Office. An abbreviated version is printed in this issue.

American Journal
of Public Health
1015 Fifteenth Street, NW
Washington, DC 20005

APHA
American Public Health Association

May 1991, Vol. 81, No. 5 ISSN: 0090-0036 Established 1911

Articles

576 Preventing Homicide: An Evaluation of the Efficacy of a Detroit Gun Ordinance *P. W. O'Carroll, C. Loftin, J. B. Waller, Jr., D. McDowall, A. Bukoff, R. O. Scott, J. A. Mercy, and B. Wiersema*

582 Cervical Papillomavirus Infection and Cervical Dysplasia in Hispanic, Native American, and Non-Hispanic White Women in New Mexico *T. M. Becker, C. M. Wheeler, N. S. McGough, S. W. Jordan, M. Dorin, and J. Miller*

587 Urolithiasis in Tennessee: An Occupational Window into a Regional Problem *M. J. Thun, and S. Schober*

592 Benzodiazepine Anti-anxiety Agents: Prevalence and Correlates of Use in a Southern Community *M. Swartz, R. Landerman, L. K. George, M. L. Melville, D. Blazer, and K. Smith*

597 The Influence of Three Mass Media Campaigns on Variables Related to Adolescent Cigarette Smoking: Results of a Field Experiment *K. E. Bauman, J. LaPrelle, J. D. Brown, G. G. Koch, and C. A. Padgett*

605 A Comparison of Injury Death Rates in China and the United States, 1986 *G. Li and S. P. Baker*

610 Comparison of Two Growth Charts in Lesotho: Health Workers' Ability to Understand and Use Them for Action *M. T. Ruel, D. L. Pelletier, J.-P. Habicht, J. B. Mason, C. S. Chobokoane, and A. P. Maruping*

Public Health Briefs

633 Hyperendemic Urban Blastomycosis *A. C. Manetti*

637 Use of a Medical Center's Computerized Health Care Database for Notifiable Disease Surveillance *M. Watkins, S. Lapham, and W. Hoy*

639 Physical Activity on the Job and Cancer in Missouri *R. C. Brownson, J. C. Chang, J. R. Davis, and C. A. Smith*

643 Vietnam Era and Vietnam Combat Veterans among the Homeless *R. Rosenheck, P. Gallup, and C. A. Leda*

646 Determinants of Late Stage Diagnosis of Breast and Cervical Cancer: The Impact of Age, Race, Social Class, and Hospital Type *J. Mandelblatt, H. Andrews, J. Kerner, A. Zauber, and W. Burnett*

Journal Departments

549 Masthead

550 Advertisers' Index

559 Information for Authors

567 Erratum

654 Notes from the Field

658 Letters to the Editor

661 Book Corner

665 Job Opportunities

American Journal of Public Health

American Public Health Association

The American Journal of Public Health, **published monthly, is the official Journal of the American Public Health Association, 1015 Fifteenth Street, NW, Washington, DC 20005; (202) 789-5600. APHA is a nongovernmental professional society founded in 1872, representing all disciplines and specialties in public health.**

APHA OFFICERS
C. William Keck, MD, MPH, *President*
Myron Allukian, Jr., DDS, MPH,
Immediate Past President
Joyce C. Lashof, MD, *President-Elect*
James M. Dunning, DDS, MPH,
Vice-President (USA)
Dr. Robert G. Evans, *Vice-President (Canada)*
Dr. Hernán Delgato, *Vice-President (Latin America)*
Caswell A. Evans, Jr., MD, MPH
Treasurer
Shirley Randolph, MSPH, *Chair, Executive Board*
George E. Hardy, Jr., MD, MPH, *Speaker, Governing Council*
William H. McBeath, MD, MPH,
Executive Director

EXECUTIVE BOARD
Shirley Randolph, MSPH (1991), *Chair*
Marilyn Aguirre-Molina, MS, EdD (1993)
Myron Allukian, Jr., DDS, MPH,
Immediate Past President
Molly Joel Coye, MD, MPH (1991)
Brian A. Dobrow, MPH (1992)
Caswell A. Evans, Jr., DDS, MPH,
Treasurer
Eugene Feingold, PhD (1991)
Kristine M. Gebbie, BSN, MN (1994)
Janet Heinrich, MPH, DrPH (1993)
Margaret H. Jordan, RN, MPH (1992)
C. William Keck, MD, MPH, *President*
Joyce C. Lashof, MD, *President-Elect*
Barry S. Levy, MD, MPH (1993)
Allan Rosenfield, MD (1994)
Reed V. Tuckson, MD (1994)
Frances M. Veverka, MPH, RS (1992)

EX OFFICIO
William H. McBeath, MD, MPH,
Executive Director
Patricia P. Evans, MPH, *Chair, Action Board*
George Pickett, MD, MPH, *Chair, Program Development Board*
George E. Hardy, Jr., MD, MPH, *Speaker, Governing Council*

The American Journal of Public Health, published monthly, is the official Journal of the American Public Health Association, Inc., 1015 Fifteenth St., NW, Washington, DC 20005. Founded in 1872, the American Public Health Association is organized to protect and promote personal and environmental health by exercising leadership in the development and dissemination of health policy. Representing all disciplines and specialties in public health, APHA is the largest public health association in the world, with an aggregate membership of 50,000, including its state and local affiliates.

Manuscripts, Correspondence, and Letters to the Editor: Send to Michel A. Ibrahim, MD, PhD, Editor, *American Journal of Public Health,* 1015 Fifteenth St., NW, Washington, DC 20005.

Information on Manuscript Preparation is published in this Journal in January. The Journal publishes reports of original research, demonstrations, evaluations, and other articles covering current aspects of public health. Manuscripts are accepted for consideration with the understanding that they, or their essential substance, have not been published previously or accepted for publication and are not under consideration elsewhere.

Subscription Price is $80/year US; $120/year international. Single copies $10 US, $14 international (surface delivery), $22 international (air delivery). Prepaid only.

Postal Information: Second class postage paid at Washington, DC, and additional mailing offices. Postmaster: Send address changes to American Journal of Public Health, 1015 Fifteenth St., NW, Washington, DC 20005. AJPH allotted $40; TNH allotted $6 of APHA membership dues. Information on individual membership appears elsewhere in this Journal, or contact: Director, Membership Services, at APHA headquarters.

Advertising Sales: Mary Beth Kammann, Advertising Manager, APHA, 1015 Fifteenth St., NW, Washington, DC 20005; (202) 789-5668 or 5600. Advertising is accepted with the understanding that products and services are professionally related and meet the ethical standards of public health practice. Acceptance by the Journal does not indicate or imply endorsement by the Journal of APHA.

Reprints: Request single reprints of articles published in the Journal from the authors at the address given in the footnote on first page of published article. For cost information on 100 or more reprints, contact: Marilyn Butler, AJPH Reprints, APHA, 1015 Fifteenth St., NW, Washington, DC 20005; (202) 789-5649.

Back Issues of the Journal: A limited inventory of back issues and volumes of the journal is available at current subscription rates plus handling charge. All orders under $35 must be prepaid. Vols. 1 to present are also available on microfilm.

Copyright © 1991 by the American Public Health Association, Inc. All material subject to this copyright may be photocopied for the noncommercial purposes of scientific or educational advancement. Opinions expressed by authors of articles summarized, quoted, or published in full in this Journal represent the opinions of the authors and do not necessarily reflect the official policy of the American Public Health Association or the institution with which the author(s) is (are) affiliated, unless so specified.

Any report, article, or paper prepared by employees of the US Government as part of their official duties is, under the Copyright Act, a "work of the United States Government" for which copyright protection under Title 17 of the US Code is not available. However, the Journal format is copyrighted and pages may not be photocopied without permission of the American Public Health Association, except in limited quantities for educational purposes, as stated above.

Copying done for other than personal or internal reference use—such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, or for resale—without the expressed permission of the American Public Health Association is prohibited. Requests for special permission or bulk orders should be addressed to APHA headquarters.

The American Journal of Public Health is indexed in whole or in part in these data bases: Bibliographic Index of Health Education Periodicals, Biological Abstracts, Biology Digest, BRS Bibliographic Retrieval Service Inc., Chemical Abstracts, Cumulative Index to Nursing & Allied Health Literature, Environmental Information Center Inc., Environmental Periodicals Bibliography, Excerpta Medica, Grateful Med, Hospital Literature Index, Hospital Management Review, Human Resources Abstracts, Index Medicus (Abridged), MEDLARS, MEDSOC, Nuclear Science Abstracts, Physical Education Index, Public Affairs Information Service, Public Health Reviews, Safety Science Abstracts, Sage Family Studies Abstracts, Science Citation Index, and Social Work Research and Abstracts.

of Health for All international conference. La Habana 1983; Ministerio de Salud Pública:25–36.

27. De Quesada Ramírez E, Galbán García, Cádiz Lahens A: SIDA: infección-enfermedad por el virus de la immunodeficiencia humana. La Habana 1987; Editorial:Científico-Técnica.

28. Marin BV: AIDS prevention in non-Puerto Rican Hispanics. Washington, DC: Govt Printing Office 1990;35–52. Pub. No. 90-1627.

29. Peterson J, Marín G: Issues in the prevention of AIDS among Black and Hispanic men. Am Psychol 1988; 43:871–877.

30. Tesh S: Health education in Cuba: A preface. Int J Health Serv 1986; 16(1):87-104.

31. Collinshaw N, Mulligan L: Recent trends in tobacco consumption—Canada and other countries. JAMA 1984; 252:23–24.

Erratum

In: Smith DB, Telles JL: Discharges against medical advice at regional acute care hospitals. Am J Public Health 1991; 81:212–215.

All of the column headings in Table 4 (which appeared on page 214) got shifted to the left one column. A corrected version of Table 4 is printed below:

TABLE 4—Percentage of Patients Discharged AMA, by Hospital Type and Major Diagnostic Category

	Teaching Hospitals								
	Suburban		City		Community				
Major Diagnostic Category	Major	Minor	Major	Minor	Suburban	City	Medical School	Other Specialty	Average
1 Nervous system	0.5	1.1	1.3	1.6	1.0	2.1	1.1	0.0	1.1
2 Eye	0.0	0.0	0.7	0.6	0.1	0.7	0.2	0.0	0.2
3 Ear, nose, and throat	0.0	0.5	0.3	0.4	0.1	0.8	0.4	0.0	0.2
4 Respiratory system	0.6	1.0	1.1	1.3	0.5	2.6	1.2	0.1	1.0
5 Circulatory system	0.6	0.7	1.2	1.0	0.7	2.0	0.8	0.1	1.0
6 Digestive system	0.4	0.6	0.9	0.9	0.5	1.7	0.7	0.0	0.7
7 Hepatobiliary system and pancreas	0.7	0.7	1.8	0.7	1.0	3.7	1.3	0.0	1.4
8 Musculoskeletal system	0.2	0.2	0.6	0.7	0.3	1.1	0.4	0.1	0.5
9 Skin, subcutaneous tissue	0.5	1.1	2.2	1.5	0.5	3.5	1.5	0.0	1.4
10 Endocrine, nutritional and met.	0.5	0.8	1.5	1.1	0.8	2.5	0.8	0.1	1.1
11 Kidney and urinary tract	0.3	0.6	0.8	0.8	0.6	1.2	1.3	0.0	0.8
12 Male reproductive system	0.3	0.1	0.4	0.7	0.3	0.7	0.5	0.4	0.4
13 Female reproductive system	0.0	0.2	0.6	0.3	0.1	0.9	0.4	0.0	0.4
14 Pregnancy, childbirth	0.1	0.0	0.4	0.1	0.2	0.3	0.5	0.0	0.3
15 Newborns and neonates	0.1	0.0	0.0	0.1	0.0	0.0	0.1	0.1	0.1
16 Blood, blood forming and imun.	0.3	2.2	1.6	2.2	1.3	5.2	2.2	0.0	1.7
17 Myeloproliferative, malignancy	0.1	0.0	0.4	0.9	0.1	0.5	0.3	0.1	0.2
18 Infections and parasitic	0.5	0.0	1.5	1.7	0.7	2.8	1.3	0.3	1.1
19 Mental	4.3	4.2	5.0	3.7	7.9	5.3	7.9	1.6	6.4
20 Alcohol/drug use	7.1	14.4	13.4	7.4	12.3	18.7	7.1	0.0	14.6
21 Injuries, poisonings and toxic	2.7	4.9	4.1	6.0	2.6	7.9	1.6	0.2	3.5
22 Burns	0.4	3.6	1.7	1.0	1.0	2.9	3.3	0.0	1.2
23 Factors influencing services	0.9	1.0	0.6	1.4	0.6	3.6	0.3	0.4	0.7
24 Other	0.0	0.0	2.6	0.0	2.0	2.5	0.5	0.0	0.8
Average	0.55	0.99	1.37	1.00	0.94	3.49	1.0	0.07	1.20

Effects of an Intervention Program on AIDS-Related Drug and Needle Behavior among Intravenous Drug Users

ABSTRACT

Background: Behaviors which entail high risk for human immunodeficiency virus (HIV) infection among intravenous drug users can be significantly reduced through educational intervention.

Methods: The educational intervention was conducted by a health educator in a one-on-one format and provided information about acquired immunodeficiency syndrome (AIDS) and strategies for reducing the risk of infection. Risk was assessed prior to the intervention and was compared to a follow-up assessment obtained approximately three months later (n = 322).

Results: The percentage who reported using drugs intravenously decreased from 92.2 to 70.5, and the percentage who reported sharing syringes dropped from 67.4 to 24.3. Reductions of these and other high-risk behaviors were detected across various demographic subgroups, and analyses show that the impact of the intervention endured for several months.

Conclusions: The findings suggest that intravenous drug users conform to a model of rational choice when confronted with accurate information about the devastating consequences of HIV infection. (*Am J Public Health* 1991;81:568–571)

Richard C. Stephens, PhD, Thomas E. Feucht, PhD, and Shadi W. Roman, PhD

Introduction

Intravenous drug users (IVDUs) constitute the second largest group of persons with acquired immunodeficiency syndrome (AIDS) in the United States and serve as a major conduit of the human immunodeficiency virus (HIV) to other groups, including non-drug-using sex partners and their children.[1,2] Although interviews with successive waves of IVDUs entering drug treatment have suggested some stabilization of HIV seroprevalence as well as some reduction in high-risk behaviors such as sharing needles and the frequency of injection,* these patterns may simply reflect changes in the composition of successive cohorts of IVDUs. Based on repeated interviews with a sample of IVDUs, panel studies have demonstrated some reduction in the frequency of injection and the frequency of sharing needles,[3-5] but the extent to which the reduction of high-risk behavior is directly responsible for the apparent stabilization of seroprevalence rates remains uncertain.[6]

In the past few years, innovative risk-reduction interventions for IVDUs have been developed. Quasi-experimental studies used to evaluate the impact of these interventions have shown risk reduction among IVDUs including a decrease in the frequency of injecting drugs** and an increase in the use of bleach to disinfect shared needles.[7]

Quasi-experimental designs are not without drawbacks. Precautions must be taken to guard against small sample size and low follow-up rate. Since the design may not allow comparisons to any type of control group, other techniques must be used to resolve questions of internal validity. Furthermore, while interviews obtained at as few as two points in time provide the researcher with no direct means of confirming the stability of the change over time, certain data analytic techniques can be used to explore the issue of stability. Each of these drawbacks is addressed in this report in which findings are presented from a quasi-experimental evaluation of an intervention program designed to reduce the chance of HIV infection among IVDUs.

Methods

Subjects

IVDUs recruited between February 1988 and August 1989 for an AIDS education and intervention research program were paid a nominal fee to participate in an initial assessment interview, an education

---

*Watters, JK: Preventing human immunodeficiency virus contagion among intravenous drug users: The impact of street based education on risk behavior. Presented at the III International Conference on AIDS, Washington, DC, June 2, 1987.

**Sufian M, Friedman SR, Goldsmith D, Des-Jarlais DC, Delana D: Risk reduction after out-reach intervention amongst intravenous drug users. Presented at the V International Conference on AIDS, Montreal, Canada, June 1989.

From the Department of Sociology, Cleveland State University (Stephens, Feucht) and the Cuyahoga County Drug Abuse Services Board, Cleveland (Roman). Address reprint requests to Richard C. Stephens, Department of Sociology, Cleveland State University, Rhodes Tower, Rm 1724, Euclid Avenue at East 24th Street, Cleveland, OH 44115. This paper, submitted to the Journal April 23, 1990, was revised and accepted for publication September 19, 1990.

TABLE 1—Needle and Drug-Related Behavior, Pretest/Posttest Comparisons Two Months Prior to Interview†

	n	Pretest	Posttest	Difference (95% CI)
Needle Behavior				
Scale means				
Using others' works	317	2.28	1.61	43.1 (36.0, 50.2)
Cleaning with bleach	315	1.86	1.87	—
% Sharing works	304	67.4	24.3	43.1 (36.0, 50.2)
% Using others' works	317	92.2	67.1	25.1 (19.4, 30.8)
% Sharing cooker	305	70.3	20.3	50.0 (44.5, 55.5)
% Using drugs IV	322	92.2	70.5	21.7 (16.1, 27.3)
% Cleaning with bleach	278	33.5	62.2	28.7 (21.0, 36.4)
Drug Behavior				
Means				
Number of drugs used	322	4.53	3.30	1.23 (1.13, 1.33)
Number of drugs used IV	322	2.24	1.43	0.81 (0.70, 0.92)
Heroin IV scale	278	4.24	3.32	0.92 (0.77, 1.07)
Cocaine IV scale	311	4.43	3.67	0.76 (0.63, 0.89)
Heroin use scale	322	3.49	2.15	1.34 (1.16, 1.52)
Cocaine use scale	322	4.47	3.27	1.20 (1.04, 1.36)
Speedball use scale	322	1.91	1.06	0.84 (0.70, 0.98)
% using heroin	322	65.5	45.7	25.9 (18.4, 33.4)
% using heroin IV	278	74.5	50.3	24.2 (16.4, 32.0)
% using cocaine	322	83.9	67.2	16.7 (10.8, 22.6)
% using cocaine IV	311	80.4	60.5	19.9 (13.8, 26.0)
% using speedball	322	40.7	27.3	13.4 (6.2, 20.6)

†The sample n varies from 322 due to missing values.

and intervention session, and a follow-up assessment begun at three months after the intervention. Pretest and posttest data were obtained on 402 subjects (60.7 percent of IVDUs from whom pretest assessments were obtained). Approximately 90 percent of the 402 subjects were street addicts not currently in treatment, and the remainder were new admissions to methadone treatment centers. The sample was predominantly male, predominantly Black, and had a median age of 36 years. Eighty subjects are not included in the analyses presented here because they were not considered to be at risk of HIV infection during the time between intervention and posttest (typically because of extended hospitalization, enrollment at a residential drug treatment facility, or incarceration) or because they participated in an alternative intervention. Most subjects were reinterviewed between three and five months after the intervention. However, because some subjects were hard to locate, the amount of time between intervention and posttest interview ranged from 36 to 448 days.

Risk Factor Measures

Pretest and posttest interviews employed multiple measures of two risk factors for HIV infection pertinent to IVDUs. Measures of risk related to needle behavior included sharing works, sharing a cooker, using drugs intravenously, using others' works, cleaning used works with bleach (each recorded as a dichotomy indicating whether the respondent had ever engaged in the behavior), and two scales measuring the frequency of using others' works and cleaning used works with bleach (where 1 = never, 2 = rarely, 3 = 25 percent of the time, 4 = 50 percent of the time, 5 = 75 percent of the time, and 6 = always). Measures of risk associated with more general issues of drug use included: number of drugs used, number of drugs used intravenously, scale scores of heroin use, cocaine use, and speedball use (where the scale is 0 = never, 1 = one to three times a year, 2 = monthly, 3 = one to three times a month, 4 = weekly, 5 = two to three times a week, 6 = four to six times a week, 7 = daily, 8 = two to three times a day, and 9 = four or more times daily), frequency of intravenous use of heroin, intravenous use of cocaine, and intravenous use of speedball (each recorded as the percent of the time that the drug is taken *intravenously* using the percentage scale described above). In addition, each of the variables pertaining to frequency of use or level of intravenous use was recorded as a dichotomy (never/at least once). All questions in the interview regarding these risk behaviors made reference to the two months immediately prior to assessment.

Intervention

The purpose of the quasi-experimental design (pretest-treatment-posttest) was to evaluate the impact of the educational intervention program. This intervention, presented in a one-on-one format by a professionally trained health educator, typically lasted from 45 minutes to an hour and was composed of four modules. The first module presented basic information about the HIV, common modes of transmission, and the progression of the disease. Because the seropositivity rate among IVDUs in Cleveland is quite low (estimates range from 3 to 6 percent based on several studies of selected groups of intravenous drug users conducted in Cleveland by the Cuyahoga County Drug Abuse Services Board) and consequently few of our clients had any immediate acquaintance with AIDS, this module centered on a segment from the film "AIDS: A Bad Way to Die,"*** produced by prisoners who were dying of AIDS. The second and third modules focused on reducing risks associated with sexual behavior and drug behavior. Condom use was demonstrated, and the relative risks of various sexual practices were discussed. Subjects were advised to modify their drug behavior by halting their use of drugs, not using drugs intravenously, not sharing works, or (as a defense of last resort against HIV infection) cleaning works when sharing with others and were shown how to clean a needle and syringe with bleach. Each subject was provided with a kit of materials including a bottle of bleach, condoms, and various brochures about AIDS. In the final module, subjects were told about the HIV antibody test and what both positive and negative results meant and were encouraged to take advantage of the anonymous blood testing provided as part of the project.

Evaluation of the intervention focused on three research questions:

- Do comparisons of pretest and posttest scores of risk behaviors provide evidence that the intervention was successful in reducing the level of risk for HIV infection among the respondents?
- In the absence of a true control group, can the impact of the intervention be assessed using a non-equivalent comparison group?
- Did the intervention have a sustained effect?

***A production of the Taconic Video Center, Taconic Correctional Facility, Taconic, NY, 1986.

TABLE 2—Assessment of Duration of Impact of the Intervention: Mean Values on Risk Behaviors for Different Pretest-Posttest Intervals

Length of Pretest-Posttest Interim	Group I ≤ 111 days n: 101	Group II 112–154 days n = 101	Group III ≥ 155 days n = 101	F-ratio ($p < 0.05$)
Needle Behavior				
Scale Means				
Using other's works	1.65	1.75	1.44	—
Cleaning w/bleach	1.70	2.12	1.80	—
% Using other's works	58.3	77.6	65.4	12.051
% Sharing works	19.6	29.0	20.6	—
% Sharing cooker	26.6	45.3	28.1	—
% Using drugs-IV	65.7	80.4	65.4	7.519
% Cleaning w/bleach	96.4	100.0	94.2	—
Drug Behavior				
Scale Means				
# Drugs used	3.11	3.57	3.22	—
# Drugs used-IV	1.21	1.69	1.39	3.987
Heroin use scale	1.66	2.36	2.45	—
Heroin IV scale	2.56	3.33	3.37	4.090
Cocaine use scale	3.41	3.60	2.82	—
Cocaine IV scale	3.59	3.99	3.19	3.229
Speedball use scale	.34	.50	.47	—
% Using heroin	35.2	51.4	50.5	7.196
% Using heroin-IV	32.4	53.3	50.5	11.139
% Using cocaine	66.7	75.7	60.7	—
% Using cocaine-IV	55.6	70.1	50.5	9.16
% Using speedball	21.3	31.8	29.0	—

Results

Six of the seven needle behavior risk behaviors decreased in frequency at the posttest (Table 1). A major reduction was obtained in the percentage who reported sharing their works, and a considerable increase was obtained in the percentage who reported cleaning works with bleach. All 12 measures of risk associated with general drug behavior recorded significant risk reduction. No significant differences in risk reduction were found between IVDUs newly enrolled in treatment and those not in treatment, or between gender or ethnic groups. Additional analyses revealed no systematic variation among groups of IVDUs in terms of knowledge about AIDS at the time of pretest.

Comparison of Experimental and Control Groups

Since a true control group was ethically unacceptable, a non-equivalent control group was constructed composed of individuals who were interviewed for the pretest at approximately the same time as those interviewed for the posttest. For 19 of the 21 measures of risk associated with needle behavior, the "experimental" (posttest) group reported significantly lower levels of risk than the "control" (contemporaneous pretest) group. Similar contrasts were found in reduction of risk associated with general drug behaviors. (Details of these comparisons are available from the authors.)

Duration of the Impact

The important question of whether the intervention had a lasting impact on reducing high-risk behavior is difficult to answer directly since interviews were conducted at only two points in time for each subject. Fortuitously, indirect evidence can be obtained using the variation in the length of time between the intervention and the posttest interview. Subjects were divided into three equal-sized groups: those interviewed within 111 days of the intervention, those interviewed between 112 and 154 days following the intervention, and those interviewed more than 154 days after intervention. Comparisons of these groups in terms of the magnitude of risk reduction are presented in Table 2. While the results do not clearly establish the stability of the intervention's impact, neither do they suggest a significant decay over time. As in the previous analyses, a greater impact was observed for needle-related behaviors than for general drug use behaviors.

Discussion

The data demonstrate that significant changes occurred between pretest and posttest in needle and drug-related behaviors of intravenous drug users, and comparisons of experimental groups with contemporaneous non-equivalent control groups suggest that these changes are at least partly attributable to the intervention. In addition, the data indicate that the impact of the intervention endured for up to one year. Finally, while reductions were detected in a broad range of behaviors associated with a high risk for HIV infection, analyses suggest that the intervention had a greater impact upon specific needle-related behaviors (such as sharing works or cleaning works) than upon more general drug-related behaviors (such as frequency of heroin or cocaine use.) All of these effects were achieved across gender and ethnic groups. These data replicate many of the findings of other earlier panel studies.[3,4,7]

Several specific limitations of the study should be noted:

- Measures of risk are based exclusively on self-report. A number of studies indicate that addicts in research projects are usually honest respondents,[8–10] and interviewers were trained to elicit the subjects' confidence. The hour-long interview was fairly complicated making misrepresentations difficult to maintain. Moreover, the variability in findings in this report (and the generally less favorable impact of the intervention upon sexual behavior to be reported elsewhere) convinces us that our data are generally reliable.
- The use of convenience sampling complicated by self-selection bias and attrition weakens generalizations to a population. Although it does not completely dispel concerns about sample bias, a comparison of dropouts and those subjects who completed the study revealed almost no significant differences in terms of pretest risk behaviors, gender, ethnicity, or age. (These data are available from the authors.) This suggests that the final study sample adequately reflects the original sample of all subjects at pre-test.
- The lack of a true control group lessens the extent to which issues of internal validity can be adequately addressed.[11]

Despite these shortcomings, these findings suggest that many addicts act in a rational fashion when faced with important choices regarding behaviors which are directly AIDS-related. It appears that the educational intervention was successful because it sensitized the subjects to the realities of AIDS, established AIDS as a real threat to them, and then immediately armed them with knowledge and materials (bleach, condoms, and assorted literature) to assist them in their own risk-reduction efforts. Levels of drug use seem somewhat resistant to intervention in this sample. However, when faced with the prospect of disease and death from AIDS, many addicts seemed willing to modify their behavior—particularly the ways in which needles and related paraphernalia are used—in order to minimize the chance of infection. ☐

Acknowledgments

This research was sponsored by a grant from the National Institute on Drug Abuse (RO1-DA05151). The authors would like to thank Brian Gibbs for his assistance in conducting the statistical analyses. The authors also wish to express appreciation to William C. Bailey, PhD, Richard Bingham, PhD, and William Morgan, PhD, for their helpful comments on an earlier draft of this paper.

References

1. Centers for Disease Control: HIV/AIDS Surveillance Report. Atlanta: CDC. July 1990; 1–16.
2. DesJarlais DC, Friedman S: The psychology of preventing AIDS among intravenous drug users: A social learning conceptualization. Am Psychol 1988; 48:865–870.
3. Friedman SR, DesJarlais DC, Sotheran JL: AIDS health education for intravenous drug users. Health Educ Q 1986; 13:383–393.
4. Robertson JR, Skidmore CA, Roberts JK: HIV Infection in intravenous drug users: A follow-up study indicating changes in risk-taking behavior. Br J Addict 1988; 83:387–391.
5. Coutinho RA: Epidemiology and prevention of AIDS among intravenous drug users. J AIDS 1990; 3:413–416.
6. DesJarlais DC, Friedman SR, Novick DM, *et al.* HIV-1 infection among intravenous drug users in Manhattan. JAMA 1989; 261:1008–1012.
7. Centers for Disease Control: Coordinated community programs for HIV prevention among intravenous-drug users—California, Massachusetts. MMWR 1989; 38:369–374.
8. Stephens RC: The truthfulness of addict respondents in research projects. Int J Addict 1972; 7:549–558.
9. Maddox JF, Desmond DP: Reliability and validity of information from chronic heroin users. J Psychiatric Res 1975; 12:87–95.
10. Amsel Z, Mandell W, Matthias L, Mason C, Hocherman I: Reliability of self reported illegal activities and drug use collected from narcotics addicts. Int J Addict 1976; 11:325–336.
11. Cook TD, Campbell DT: Quasi-Experimentation: Design and Analysis Issues for Field Settings. Boston: Houghton Mifflin, 1979.

Harbor-UCLA Selected for Psychiatry Research Center

The National Institute of Mental Health (NIMH) recently approved the establishment of the nation's first mental health research center, the Research Center of the Psychobiology of Ethnicity, at the Harbor-UCLA Medical Center in Torrance, California. The $1.5 million grant will be administered by Harbor-UCLA's Research and Education Institute.

Alan I. Leshner, PhD, Acting Director of NIMH, said, "Although mental illness can strike anyone regardless of race or cultural origin, there appear to be some very important differences in the ways people of different ethnic backgrounds respond to medications in the treatment of depression, schizophrenia and other serious mental disorders. The research undertaken by the Harbor-UCLA Hospital Center will greatly enrich our understanding of the psychobiological bases of mental disorders and their treatment, and help ensure that every patient of every ethnic background receives appropriate treatment."

Center director Keh-Ming Lin, associate professor of psychiatry at Harbor-UCLA, described the focus of the center's programs as multidimensional and designed to serve the multiethnic populations of the community. The center will study the role that race and ethnicity play in the differing responses to treatments of serious mental illnesses. Drugs used in the management of mental depression, mania, and schizophrenia have been shown to have differing impacts, and to reach different levels in the blood and the brain, with ethnicity being a significant variable.

In addition to the study of biological issues and their impact on mental illnesses in people of differing ethnicity, the center will also study how cultural factors influence the evaluation and care of patients, and how they impact on the willingness of patients and families to make use of treatments and support programs found useful in mental health care.

Lin explained that "a very serious problem exists among minority patients and their families because they rarely make use of the self-help and patient/family support groups like the California Alliance for the Mentally Ill which provide so much assistance to those who do participate."

Lin and his colleagues are currently involved in a research project with Hispanic patients and families to identify ways to make treatment programs and self-help group activities more culturally acceptable and to increase access for these individuals to such programs. Other studies underway focus on the effect of mental illnesses on Asian, Black, and Native American patients and their families.

For additional information, contact the Office of Scientific Information, National Institute of Mental Health, 5600 Fishers Lane, Room 15-105, Rockville, MD 20857; (301) 443-4536.

Adult Survivors of Childhood Sexual Abuse and Subsequent Risk of HIV Infection

Sally Zierler, DrPH, Lisa Feingold, MSPH, Deborah Laufer, MSPH, Priscilla Velentgas, Ira Kantrowitz-Gordon, ScB, and Kenneth Mayer, MD

ABSTRACT

Background: Epidemiologic description of long-term adverse health effects of childhood sexual abuse is lacking, despite estimates that perhaps 30 percent of adults have experienced sexual assault in childhood.

Methods: In an adult cohort enrolled to investigate causes of transmission of human immunodeficiency virus, we identified current behaviors affecting risk of infection that were associated with a history of early sexual abuse. One hundred and eighty-six individuals provided information on the occurrence of abuse and subsequent sexual and drug using activities.

Results: Approximately half of the women and one-fifth of the men reported a history of rape during childhood or adulthood. Twenty-eight percent of the women and 15 percent of the men recalled that they had been sexually assaulted during childhood. People who reported childhood rape compared with people who did not were four times more likely to be working as prostitutes (90 percent confidence interval = 2.0, 8.0). Women were nearly three times more likely to become pregnant before the age of 18 (90% CI = 1.6, 4.1). Men who reported a history of sexual abuse had a twofold increase in prevalence of HIV infection relative to unabused men (90% CI = 1.0, 3.9).

Conclusions: The disturbing prevalence of early sexual abuse and its possible health-related consequences call for prompt and routine investigation of sexual abuse histories. Identification of sexual victimization may be an important component for management of risk factors for human immunodeficiency virus. (*Am J Public Health* 1991;81:572–575)

Introduction

The social sciences have become increasingly concerned with the prevalence and psychosocial consequences of sexual abuse in childhood.[1-10] Prevalence studies in communities of free-living adults estimate that 6 to 62 percent of women and 3 to 31 percent of men were sexually abused before the age of 18.[11] The range in these estimates is a function of the specificity of the definition of sexual abuse and the age definition for childhood. When the definition is limited to children under 14 years of age who reported physical contact of a sexual nature, estimates range from 28 to 36 percent.[3] In 1985, the American Humane Association reported 123,000 cases of childhood sexual abuse during that year alone, and yet this figure is likely to be a gross underestimate.[11]

Childhood sexual abuse is a medical problem that requires careful diagnosis and follow up for trauma,[12] pregnancy,[13] disease transmission, including human immunodeficiency virus (HIV) infection,[13-16] and acute psychological morbidity.[11-13,17,18] Two behavioral outcomes that are associated with sexual abuse in childhood are chemical substance abuse and prostitution.[10,11] These risk-taking behaviors have health-related consequences that theoretically could include any diseases resulting from intravenous drug use or unprotected sexual activity with multiple partners. Infection from HIV would be associated with childhood sexual abuse if such abuse predisposes children to drug using or sexual behaviors that involve contact with bodily fluids infected with this virus.

An explicit goal of our study of determinants of heterosexual transmission of HIV infection in the Rhode Island area was to learn if a history of sexual abuse is a risk factor for subsequent HIV infection in adulthood. We hypothesize that adults who were sexually abused as children are more likely to participate in activities that increase their risk of exposure to HIV.

Methods

Study Population

All women and men at least 18 years old, residing or attending school in southeastern New England, currently or recently sexually active with someone of the opposite sex, and who signed an informed consent statement to participate in the research protocol were eligible to be in the study. Potential study volunteers contacted the study clinic as a result of the recommendation from a health care provider (provider-referred) or because of interest stimulated by a large scale publicity campaign (self-referred).

From the population of 567 women and men who enrolled in the testing and counseling program between March 1988 and October 1989, 393 people were invited to participate in the longitudinal study because they had a nonzero risk of acquiring or transmitting HIV infection (either because they had multiple sexual partners, were themselves HIV-infected, or had

---

From the Department of Community Health, Division of Biology and Medicine, Brown University, all authors except Laufer who is with the Infectious Disease Division at Memorial Hospital of Rhode Island where Dr. Mayer is also affiliated. Address reprint requests to Sally Zierler, DrPH, Department of Community Health, Brown University, Box G-A405, Providence, RI 02912. This paper, submitted to the Journal May 2, 1990, was revised and accepted for publication October 10, 1990.

partners who were infected or at increased risk for HIV infection). Selection of the cohort and the decision to participate were independent of a history of childhood sexual abuse, since a history of sexual abuse was not known by the study personnel at the time of enrollment, nor had the potential participant seen or completed the questionnaire which investigated that history. There were 206 people who were eligible for the longitudinal study but refused to participate. The discontinued study population relative to the continuing study population was more likely to be male and less likely to be HIV-infected. The people who discontinued compared with those who agreed to remain were similar with respect to their age distribution, number of different sexual partners, race and ever use of intravenous drugs. Among the 187 individuals who agreed to continue in the study by signing a second informed consent statement, 186 had complete information on history of sexual assault. This population provided the information for the current study.

Exposure Information: A certified HIV counselor or nurse-clinician administered a lengthy behavioral questionnaire to study participants at the time they agreed to be in the longitudinal study. Information on the exposure of interest, a reported history of sexual abuse, was based on the question, "Have you ever been raped or forced to have sex?" People who acknowledged a history of sexual abuse further remarked on the frequency and timing of this occurrence (during childhood, adolescence, adulthood or during any combination of these time periods).

For the purpose of this analysis, individuals were defined as exposed to childhood sexual abuse if they reported a history of rape or forced sex at least once during childhood or as a teenager. Individuals were defined as not exposed if they reported no history of rape or forced sex, including during adulthood. Individuals reporting a history of first rape as an adult (n = 22) were excluded from the analyses due to our inability to determine a temporal sequence between exposure to sexual abuse and the outcomes of interest.

Outcome Information: Outcomes of interest included behaviors that may increase risk of acquiring HIV infection, as well as biologic parameters such as a history of sexually transmitted disease or current HIV infection. Information for the behavioral outcomes was obtained from the questionnaire administered at entry into the longitudinal study. Individuals provided information on their lifetime use of intravenous and non-intravenous illicit drugs, alcoholic beverages, contraceptives, and prior or current work as a prostitute. Detailed history of number of sexual partners, number of casual sexual partners, number of years of sexual activity, sexual practices, education, and pregnancy experience provided additional information on relevant outcomes.

TABLE 1—Demographic Characteristics according to Reported History of Childhood Sexual Abuse

	Child Sexual Abuse* (n = 41)	No Sexual Abuse (n = 123)	Total (n = 164)
	%	%	%
Gender			
Female	71	44	51
Male	29	56	49
Age (years)			
18–24	10	16	15
25–34	58	51	53
35–44	27	26	26
45+	5	7	6
Race			
White	76	80	79
Other	24	20	21
Referral			
Provider	54	47	49
Self	46	53	51

*Reported history of sexual abuse as a child or teenager.

TABLE 2—Prevalence of Reported Sexual Abuse according to Time of First Occurrence

	Women (n = 101)		Men (n = 85)		Total (n = 186)	
	n	%	n	%	n	%
Never sexually abused	54	53.5	69	81.2	123	66.1
Childhood	20	19.8	8	9.4	28	15.1
Teenager	9	8.9	4	4.7	13	7.0
Adult*	18	17.8	4	4.7	22	11.8

*22 individuals reporting first abuse as adults were deleted from all subsequent analyses.

Sexually transmitted disease history was obtained by self-report. HIV antibody status was determined by serologic testing by standard enzyme-linked immunoassay and Western Blot procedures using fresh blood samples that were drawn at the time of entry into the study.

All outcome measurements were created as dichotomous variables defined as the presence or absence of the outcome of interest. Examination of non-intravenous drug use and alcohol use was limited to those reporting no history of intravenous drug use because of the strong association of intravenous drug use with nonparenteral use of other drugs.

Data Analysis: Prevalence ratios provided a quantitative measure of risk between the outcome of interest and child sexual abuse. Prevalence ratios were computed as the proportion of people who had a particular outcome within the population who reported a history of child sexual abuse relative to the proportion who had this outcome within the population who reported no history of sexual abuse at any time.

Multiple logistic regression was used to examine the possible confounding effects of gender, race, and study referral source. Evaluation of confounding indicated that race and referral pattern did not materially change the coefficient for the child abuse term (for all outcomes, the coefficient for child abuse changed less than 5 percent) and therefore these potential confounders were deleted from the analysis. Table 1 lists the demographic characteristics of the cohort that was included in the analysis.

Because gender was the only measured confounder, and also because of in-

TABLE 3—Behaviors Associated with a History of Childhood Sexual Abuse

	Childhood Sexual Abuse							
	Women			Men			Overall	
Behavior	Yes (n = 29)	No (n = 54)	Prev. Ratio	Yes (n = 12)	No (n = 69)	Prev. Ratio	Prev. Ratio_{MH}**	90% Confidence Interval
Prostitution	6	4	2.8	4	3	7.6	4.0	(2.0, 8.0)
IVDU**	8	13	1.2	6	32	1.2	1.2	(.78, 1.8)
Average yearly sexual partners (>2 vs ≤2)	15	16	1.7	10	24	2.4	2.0	(1.4, 2.8)
Never/rarely use condom in last year†	21	36	1.0	5	41	0.8	1.0	(.76, 1.2)
Sex with strangers††	23	28	1.5	10	52	1.2	1.4	(1.1, 1.6)
Education (<HS vs ≥HS)	9	5	3.4	2	15	0.8	1.9	(1.0, 3.5)
Pregnancy before 18 years old	18	13	2.6	—	—	—	2.6	(1.6, 4.1)

*Prev ratio_{MH} are Mantel-Haenszel estimates of prevalence ratios pooled over gender strata.

**One woman and one man with history of child sexual abuse were deleted from analysis because temporal sequence of aga of sexual abuse and age of IV drug use not specified

†Four sexually inactive individuals deleted from analysis.

††Eight individuals deleted due to missing data.

TABLE 4—Use of Alcohol and other Drugs and Childhood Sexual Abuse among Adults with no History of Intravenous Drug Use*

	Childhood Sexual Abuse							
	Women			Men			Overall	
Drug use†	Yes (n = 20)	No (n = 41)	Prev. Ratio	Yes (n = 5)	No (n = 37)	Prev. Ratio	Prev. Ratio_{MH}**	90% Confidence Interval
Cocaine	8	20	0.8	3	20	1.1	0.9	(0.59–1.3)
Tranquilizer	9	11	1.7	2	9	1.6	1.7	(0.98–2.8)
Marijuana	19	33	1.2	4	32	0.9	1.1	(0.95–1.3)
Hallucinogens	6	14	0.9	1	10	0.7	0.8	(0.46–1.6)
Heavy alcohol	6	6	2.0	3	14	1.6	1.8	(1.0–3.3)

*22 women and 39 men intravenous drug users deleted from analysis.

**Prev ratio_{MH} are Mantel-Haenszel estimates pooled over gender strata.

†Drug use was defined as ever use of these drugs (no use was defined as never used); Heavy alcohol use was defined as usual consumption of at least one bottle of wine, five cans of beer, or one pint of hard liquor when drinking (no heavy use was typically drinking less than this amount.)

terest in describing gender-specific associations, all analyses were stratified by gender and summarized as Mantel-Haenszel summary prevalence ratios with 90 percent test-based confidence intervals.[19]

Results

Approximately one out of every two women and one out of every five men reported a history of rape or forced sex (Table 2). The proportion of women and men who reported sexual abuse during childhood or adolescence was 28 and 15 percent, respectively.

The associations for behavioral characteristics and exposure to early sexual abuse appear in Table 3. Survivors of child sexual abuse were four times more likely to report having worked as a prostitute during their lifetime than were those who reported no history of abuse. This higher prevalence of prostitution among survivors was seen among both women and men. Male survivors of abuse were nearly eight times more likely to report a history of prostitution. All of the men reporting prostitution also reported bisexual experiences. Excluding male prostitutes, bisexuality among men was not associated with childhood sexual abuse (prevalence ratio = 1.1). Additional analyses, simultaneously controlling for gender and intravenous drug use, a behavior that may lead to prostitution as a source of income for drugs, resulted in only a slight lowering of the point estimate (prevalence ratio = 3.6, 90 percent CI = 1.8, 7.3). Use of intravenous drugs was only weakly associated with a history of early sexual abuse.

Penetrative sex without a condom with a partner suspected of having HIV infection was similar among survivors of child sexual abuse and among those not abused (prevalence ratio = 1.1). No difference between the two groups existed with respect to the lack of condom use in the year prior to enrolling in the study. Survivors of sexual abuse, however, reported a 40 percent excess frequency of a history of sex with someone they did not know, and were two times more likely to have multiple sexual partners on an average yearly basis relative to people reporting no sexual abuse.

Women who survived sexual abuse had greater than a three-fold increase in risk of not completing high school than women who reported no sexual abuse. This increase was explained in part by a higher occurrence of teenage pregnancy among the women who did not finish high school. Adjustment for pregnancy prior to the age of 18 lowered the point estimate for the relation between education level and sexual abuse history from 3.4 to 1.9 (90 percent CI = 0.8, 4.3). Teenage pregnancy was more prevalent among female survivors of sexual abuse than among those reporting no history of abuse (prevalence ratio = 2.6, 90 percent CI = 1.6, 4.1).

Table 4 describes drug use associations among people with no history of intravenous drug use. Survivors of sexual abuse had a 70 and 80 percent excess of use of tranquilizers and heavy alcohol use, respectively, relative to people reporting no sexual abuse. Women were two times more likely to be heavy consumers of alcohol if they had experienced sexual abuse in childhood than women who did not report this experience.

Overall, survivors of child sexual abuse were only slightly more likely to report a history of sexually transmitted disease (gender-adjusted prevalence ratio = 1.3, 90 percent CI = 0.93, 1.9) or to have serologic evidence of HIV infection (gender-adjusted prevalence ratio = 1.3, 90 percent CI = 0.82, 2.0). However, men who reported early sexual abuse had a two-fold increase in prevalence of HIV infection relative to unabused men (90 percent CI = 1.0, 3.9). This increased prevalence of HIV infection among men who survived sexual assault was not explained by a history of intravenous drug use.

Discussion

National estimates are that one in four girls and one in six boys are sexually assaulted before the age of 18.[20] Over one-fifth of our cohort acknowledged that they had been raped or forced to have sex during childhood or adolescence. This experience was twice as frequent among women as among men.

We do not have evidence of confounding of sexual abuse effects. Our observations are consistent with other studies that have described sexual abuse to be independent of race.[4,11] Unidentified correlates of childhood sexual abuse that are independent risk factors for behaviors leading to HIV infection could explain the positive results in this study. However, we did not ask about child and adolescent experiences other than those related to sexual and reproductive experiences and age of first use of illicit drugs. Childhood sexual abuse, particularly if the perpetrator is a family member, may be accompanied by other forms of abuse and neglect.[9] Our study did not investigate characteristics of family dysfunction that may be associated with both sexual abuse and adult behaviors leading to HIV infection.

We have evidence that early sexual abuse is associated with behavioral outcomes that may be having devastating effects on the public health, particularly in relation to the HIV epidemic. In our study, sexually abused women and men were more likely to engage in sex work, to change sexual partners frequently, and to engage in sexual activities with casual acquaintances than people who were never sexually abused. Women survivors of sexual assault reported more frequent use of large quantities of alcoholic beverages and both genders used tranquilizers more frequently than individuals who had never been assaulted.

The disturbing prevalence of early sexual abuse and its possible consequences on increasing behaviors that could lead to HIV infection and other poor health outcomes have implications for medical and public health practitioners. The safer sex messages, for example, may be missing the point for people whose lives have been complicated by sexual victimization. Social scientists have urged for early identification of children and adolescents who may have been sexually assaulted because of evidence that the earlier the child can begin recovery from this trauma, the better the prognosis for normal adult functioning[3,18] The high frequency of occurrence of sexual assault is not only an immediate threat to the health of children, but may continue to disrupt development of appropriate health behaviors that prevent disease in adulthood. ☐

Acknowledgments

We are indebted to the staff of the New England Behavioral Health Study: Charles Carpenter, MD; Frances Bettencourt, Gale Frade, Beverly Levin, and Douglas Reed, nurse clinicians; David Abbott, Carlos Del Valle, Lionel Fernandez, and Norma Hardy, health educators; Lynne McCrow, senior lab technologist; Kathryn Titus, administrative assistant. And to the women and men who courageously shared their struggle and hope for this research.

This research is funded by the National Institute of Allergies and Infectious Diseases of the National Institutes of Health, Grant #R01-AI-25828.

References

1. Russell DEH: The incidence and prevalence of intrafamilial and extrafamilial sexual abuse of female children. Child Abuse Negl 1983; 7:133–146.
2. Baker AW, Duncan SP: Child sexual abuse: A study of prevalence in Great Britain. Child Abuse Negl 1985; 9:457–467.
3. Wyatt GE, Peters SD: Issues in the definition of child sexual abuse in prevalence research. Child Abuse Negl 1986; 10:231–240.
4. Wyatt GE: The sexual abuse of Afro-American and White-American women in childhood. Child Abuse Negl 1985; 9:507–519.
5. Finkel KC: Sexual abuse in children: An update. Can Med Assoc J 1987; 136:245–252.
6. Finkelhor D: Child Sexual Abuse: New Theory and Research. New York: The Free Press, 1984: 69–86.
7. Nash CL, West DJ: Sexual molestation of young girls: A retrospective survey in sexual victimization. *In:* West DJ (ed): Sexual Victimization. Brookfield, Vermont: Gower Publishing, 1985; 1–87.
8. Faller KC: Child Sexual Abuse. New York: Columbia University Press, 1988; 22–46.
9. Radbill SX: Children in a world of violence. *In:* Helfer RE, Kempe RS (eds): The Battered Child. Chicago: University of Chicago Press, 1987; 9–12.
10. James J, Meyerding J: Early sexual experience and prostitution. Am J Psychiatry 1977; 134:1381–1385.
11. Finkelhor D: The sexual abuse of children: Current research reviewed. Psychiatric Ann 1987; 17:233–241.
12. Herman-Giddens ME, Berson NL: Harmful genital care practices in children. JAMA 1989; 261:577–579.
13. American Academy of Pediatrics, Committee on Adolescence: Rape and the adolescent. Pediatrics 1988; 81:595–597.
14. Gellert GA, Durfee MJ: HIV infection and child abuse (letter). N Engl J Med 1989; 321:685.
15. Gellert GA, Mascola L: Rape and AIDS (letter). Pediatrics 1989; 83(Suppl):644.
16. Osterholm MT, MacDonald KL, Danila RN, Henry K: (letter). N Engl J Med April 16, 1987; 316:1024.
17. Herman J, Russell D, Trocki K: Long-term effects of incestuous abuse in childhood. Am J Psychiatry 1986; 143:1293–1296.
18. Kempe CH: Sexual abuse, another hidden pediatric problem: The 1977 C. Anderson Aldrich Lecture. Pediatrics 1978; 62:382–389.
19. Miettinen OS: Estimability and estimation in case-referent studies. Am J Epidemiol 1976; 103:226–235.

Preventing Homicide: An Evaluation of the Efficacy of a Detroit Gun Ordinance

Patrick W. O'Carroll, MD, MPH, Colin Loftin, PhD, John B. Waller, Jr., DrPH, David McDowall, PhD, Allen Bukoff, PhD, Richard O. Scott, James A. Mercy, PhD, and Brian Wiersema

ABSTRACT

Background: **In November 1986, a Detroit, Michigan city ordinance requiring mandatory jail sentences for illegally carrying a firearm in public was passed to preserve "the public peace, health, safety, and welfare of the people."**

Methods: **We conducted a set of interrupted time-series analyses to evaluate the impact of the law on the incidence of homicides, hypothesizing that the ordinance, by its nature, would affect only firearm homicides and homicides committed outside (e.g., on the street).**

Results: **The incidence of homicide in general increased after the law was passed, but the increases in nonfirearm homicides and homicides committed inside (e.g., in a home) were either statistically significant or approached statistical significance (p = .006 and p = .070, respectively), whereas changes in the incidence of firearm homicides and homicides committed outside were not statistically significant (p = .238 and p = .418, respectively). We also determined that the ordinance was essentially unenforced, apparently because of a critical shortage of jail space.**

Conclusions: **Our findings are consistent with a model in which the ordinance had a dampening effect on firearm homicides occurring in public in Detroit. The apparent preventive effect evident in the time series analyses may have been due to publicity about the ordinance, whereas the small nature of the effect may have been due to the lack of enforcement. (*Am J Public Health* 1991;81:576–581)**

Introduction

The homicide rate in Detroit, Michigan, was the highest among the 15 largest United States cities in all but three of the last 15 years.[1,2] In 1986, the crude homicide rate in Detroit was 59 per 100,000, the highest of any large city in the United States. As is the case nationally, most homicide victims in Detroit were killed with firearms. In 1985 in Michigan, firearm homicides accounted for 72.7 percent of the homicide-attributable premature loss of life among Blacks. Two-thirds of Michigan's homicides occurred in the Detroit area.[3]

On November 26, 1986, the Detroit City Council, responding to a wave of youth homicides, enacted an ordinance[4] that imposed a mandatory jail sentence on anyone convicted of unlawfully concealing a pistol or carrying a firearm in the city. Although the ordinance contained some technical amendments to the city ordinances regulating the carrying and selling of firearms, the key element of the ordinance was the mandatory sentence of 30 to 90 days in the Wayne County jail and a fine of $100 to $500, depending on whether or not it was a first conviction under the ordinance. The ordinance became effective on January 10, 1987.

One of the primary goals of this legislation was to improve the health of the citizens of Detroit by decreasing firearm-related injuries and deaths. It was specifically stated in the ordinance that this legislation was "necessary for the preservation of the public peace, health, safety, and welfare of the people of the City of Detroit." This ordinance may therefore be viewed as a public health intervention, and its efficacy evaluated in that context.

Underlying this ordinance is the hypothesis that, by decreasing the number of persons carrying firearms on the streets of Detroit, the incidence of firearm homicides resulting from interpersonal violence occurring "outside" (e.g., on streets, in alleys, or in parking lots) would likewise decrease. Since the ordinance does not address the issues of possessing or keeping firearms in private places (such as the home) or of carrying other weapons (such as knives) in public, the law should have its greatest effect on the incidence of firearm homicides and homicides committed in public places while having little effect on the incidence of nonfirearm homicides or on firearm homicides committed inside private residences and other buildings.

To determine whether the imposition of the mandatory jail sentences in January 1987 was associated with changes in the incidence both of homicides committed with firearms and homicides committed outside, we analyzed homicide incidence data in the City of Detroit for the years

---

From the Division of Injury Control, Center for Environmental Health and Injury Control, Centers for Disease Control, US Public Health Service, Atlanta, GA (O'Carroll, Mercy); Institute of Criminal Justice and Criminology, University of Maryland, College Park, MD (Loftin, Wiersema); Center for Prevention and Control of Interpersonal Violence, Department of Community Medicine, Wayne State University School of Medicine, Detroit, MI (Waller, Bukoff, Scott); and School of Criminal Justice, State University of New York, Albany, NY (McDowall).

Address reprint requests to Patrick W. O'Carroll, MD, MPH, Mailstop F36, Centers for Disease Control, 1600 Clifton Road NE, Atlanta, GA 30333. This paper, submitted to the Journal August 6, 1990, was revised and accepted for publication December 20, 1990.

1980 through 1987. For comparison, we also analyzed nonfirearm homicides and homicides committed inside residences and other buildings. We predicted that changes attributable to the 1987 ordinance would be reflected in the incidence of firearm homicides and homicides committed outside, but not in the incidence of nonfirearm homicides or homicides committed inside.

In addition, we evaluated the enforcement of the mandatory sentencing ordinance by the criminal justice systems in Detroit. Although one might assume that the ordinance would increase the average sentence for persons convicted of illegally carrying a firearm in Detroit, experience in Detroit and other locations indicates that mandatory sentences do not necessarily produce the expected changes in average sentences.[5-9] For a variety of reasons, judges find ways to circumvent the mandatory sentences, or discretion for sentencing moves to another point in the criminal justice system such as the prosecutor or the police. An examination of how the law was implemented is therefore essential to the interpretation of the time-series analysis. In addition, the Detroit experience is important for what it may reveal about how a criminal justice system adapts to mandatory sentencing for carrying a firearm illegally.

Methods

Evaluating Effect of the Ordinance

Homicide data for the years 1980 to 1987 were drawn from computerized Official Complaint Report (OCR) records maintained by the Detroit City Police Department. All criminal homicides were included in the analysis, including offenses coded as murder, murder (1st degree), murder (2nd degree), and manslaughter.

OCR codes were used to divide the homicides two ways: 1) those in which the victim was killed with a *firearm* (handgun; shotgun/rifle) or by *some other means* (bodily force; black jack; knife, dirk, razor/other sharp objects; blunt object; miscellaneous other); and 2) those in which the victim was killed *outside* (on street; off street, alley; playground/park; parking lot; in a vehicle; vacant lot) or *inside* (building; housing project). Four cases in which the homicide method was coded as "missing" and 73 cases coded as "unknown" were assigned to the nonfirearm series on the grounds that firearm homicides are easily recognized. The police data did not permit a determination of whether homicides in "buildings" occurred in private buildings (such as in a home) or in public buildings (such as in a bar or convenience store). Including homicides in public buildings in our *outside* series would have been preferable, since weapons used to commit such homicides would presumably have been carried "outside" to these buildings, and thus the perpetrators should theoretically have been affected by the ordinance. Nevertheless, if a substantial proportion of the homicides in buildings occurred in public buildings, this would result in a bias toward the null in our study since it would tend to dampen the hypothesized preventive effect on homicides committed outside relative to those committed inside.

Inferences about the impact of the ordinance on homicides are based on an interrupted time-series research design that contrasts the monthly incidence of certain types of homicide before and after the ordinance took effect. A change in the behavior of a particular time series at the point of intervention would provide evidence that the ordinance had an effect on that time series. To measure the size and statistical significance of time-series changes associated with the implementation of the gun ordinance, we used the intervention analysis developed by Box and Tiao based on the Box and Jenkins autoregressive, integrated, moving average (ARIMA) modeling of time series.[10,11] In general terms, the procedure starts with the development of a *noise model* to account for systematic components—trend or drift, seasonality, and autocorrelation in the time series. Modeling these systematic components to explicitly remove their effects is important because it reduces the risk that these components will be confounded with an intervention effect. This noise model essentially controls for variables which influence the homicide rates consistently over time, allowing analysts to estimate the effects of the intervention. After an appropriate noise model to control the effects of these series characteristics has been specified, an *intervention* model is added to represent the effect of the ordinance. If there is a change in the series at the point of intervention, the intervention model will measure the size of the effect.

Ideally, we would have conducted the analysis on monthly observations for each of the four time series resulting from the logical intersection of the two dimensions of interest: 1) firearm homicides in public places; 2) nonfirearm homicides in public places; 3) firearm homicides in nonpublic places; 4) nonfirearm homicides in nonpublic places. However, there were insufficient cases to permit ARIMA modeling of the time series in this manner. Therefore, we conducted two separate monthly analyses: one for firearm versus nonfirearm homicides (categories 1 + 3 versus 2 + 4) and one for public versus nonpublic homicides (categories 1 + 2 versus 3 + 4).

All estimates were computed using the backforecasting algorithm implemented in BMDP2T software.

Evaluation of Enforcement

To assess the impact of the mandatory jail sentencing ordinance on the processing of gun ordinance violators in the Detroit criminal justice system, we monitored the disposition of cases charged under the ordinance during 1987 and 1988 using data that are routinely collected by the prosecutor and the court. In addition, we conducted non-structured interviews with key people in the Detroit criminal justice system, people whose jobs involved implementing the ordinance or processing defendants charged with violating the ordinance, e.g. members of the Detroit Police Department, the Department of the Wayne County Prosecuting Attorney, the Office of the Detroit Corporate Council (city solicitor), the 36th District Court, and others. These interviews were not intended to constitute a scientific survey; rather, they were intended to supplement the statistical analysis of the disposition of offenders charged under the ordinance by allowing a description of perceptions regarding the enforcement of the ordinance by those most likely to be well informed in this regard.

Results

Findings of the Time-Series Analysis

The two sets of time series data—firearm vs nonfirearm homicides, and inside vs outside homicides—are plotted in Figures 1 and 2. The results of the interrupted time-series analyses of these data are summarized in Table 1. For each of the four series that we analyzed, the table shows the estimated size of the intervention effect (in terms of the change in the number of homicides per month associated with the intervention); the mean number of homicides per month before the intervention; the relative magnitude of the intervention effect (expressed as a percent change in the preintervention mean number of homicides per month); and the

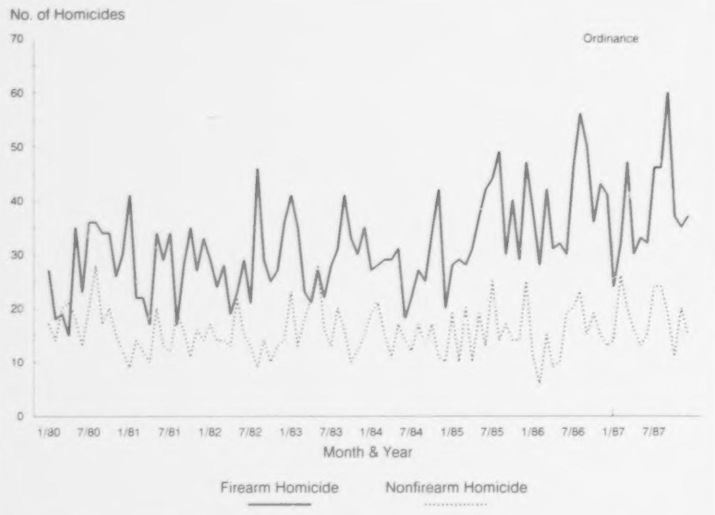


FIGURE 1—Number of Homicides in Detroit by Month and Weapon Used, 1980–87

p-value, representing the probability of a Type-I error associated with a test of the null hypothesis of a zero intervention effect. In the Appendix, we provide more complete information on the time-series analysis.

The clearest feature of the analysis, reflected in the positive estimates of the intervention effect in all four series (Table 1) is that in January 1987, as the ordinance was implemented, Detroit was experiencing a substantial increase in homicides. This is consistent with the public perception in Detroit of increasing homicide incidence. As noted above, this perception is what stimulated the Council to pass the mandatory sentencing ordinance.

Although the ordinance clearly failed to stop the surge, a comparison among the various time series of changes associated with the intervention in the mean number of homicides per month suggests that the ordinance may have had a dampening effect on the increasing incidence of homicide. Homicides committed inside (those unlikely to be affected by the ordinance) increased by 22 percent, a much greater increase than the 10 percent increase in homicides committed outside (those more likely to be affected by the ordinance). The statistical analysis shows that the increase in inside homicides is large relative to its standard error (see Appendix) and is therefore unlikely to be due to a chance fluctuation (p = .006); the increase in outside homicides, however, is small relative to its standard error and is compatible with a variation attributable to chance (p = .418). The data are therefore consistent with a model in which the incidence of homicides committed inside increased, but the incidence of homicides committed outside was unchanged.

A similar but less pronounced pattern exists for the firearm/nonfirearm contrast. The incidence of nonfirearm homicides increased 16 percent after the intervention, a change that is large relative to the standard error of the series and therefore relatively unlikely to be due to a chance fluctuation (p = .07). Although the incidence of firearm homicides increased almost as much (13 percent over the mean of the preintervention series), the change was small relative to its standard error and not statistically significant by conventional standards (p = .24). In other words, the increases in the two types of homicide most clearly targeted by the ordinance were not statistically significant; plausibly the increases observed were random fluctuations around the preintervention mean. In contrast, the increases in the two comparison series that were least likely to be influenced by the ordinance either were clearly statistically significant or approached statistical significance at the p = .05 level. The patterns are what would be expected if the Detroit gun ordinance had a dampening effect on firearm homicides and outside homicides.

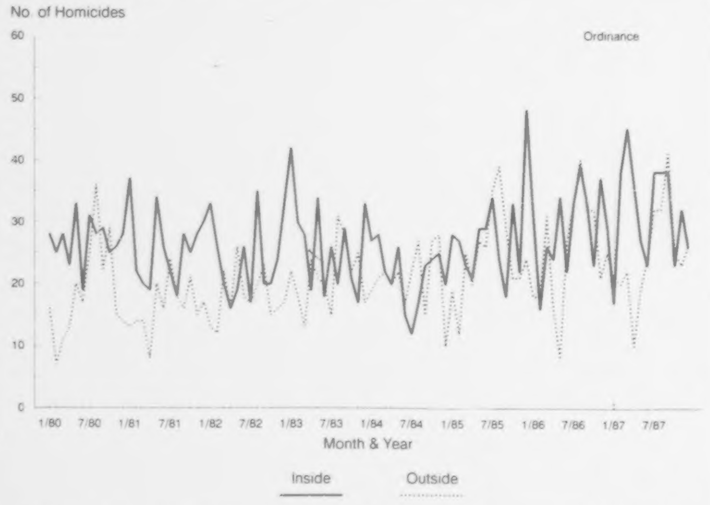


FIGURE 2—Number of Homicides in Detroit by Month and Location of Homicide, 1980–87

Enforcement of the Ordinance

The research design assumes that the ordinance resulted in the mandated change in jail sentences for illegally concealing a pistol or carrying a firearm. We determined, however, that the law did not result in any such change. The records of the 1,020 persons charged under the ordinance whose cases were processed as of early 1989: only 22 defendants (2.2 percent) were sentenced to jail and eight others (0.8 percent) were given credit for jail time served while waiting disposition of their case. These findings were confirmed informally through our interviews with key criminal justice personnel. Two years after the ordinance was passed, everyone we interviewed who was in a position to observe sentencing practices agreed that extremely few of those who were arrested for carrying concealed weapons ultimately received the mandatory sentences stipulated in the ordinance.

The administrative context in which the ordinance is enforced explains why it failed to increase the certainty or severity of sentences. Since long before the ordinance was passed, officials at the Wayne County Jail had been under court order to reduce overcrowding and improve living conditions. This order had transformed jail space into a scarce and expensive commodity. Judges had to choose who among the misdemeanants, ordinance violators, and pretrial felony defendants to incarcerate in the 1,676-bed county jail. Our informants agreed unanimously that it was impossible to enforce the mandatory sentencing provisions of the ordinance because not enough jail space was available. Given a choice between incarcerating violators of the gun ordinance versus pretrial felony defendants or other offenders who had committed more serious offenses such as violent assault or robbery, ways were found to avoid the mandatory sentencing provision of the gun ordinance.

TABLE 1—Summary of Interrupted Time-series Analysis of the Effect of a Gun Ordinance on the Incidence of Homicide, Detroit, Michigan, 1980 to 1987

Homicide Series	Estimate of Intervention Effect	Mean Number of Homicides[a]	Percentage Change in Series[b]	P-value for Test of H_0 = no change
Inside	5.798	25.95	22	.0058
Outside	2.086	20.83	10	.4179
Nonfirearm	2.524	15.56	16	.0703
Firearm	4.109	31.42	13	.2380

[a] The mean number of homicides per month in the preintervention series adjusted, where appropriate, for autocorrelation.

[b] The intervention effect as a percent of the mean of the preintervention series.

Discussion

Unquestionably, homicide is an enormous public health problem in the United States.[12] Viewed strictly from a health perspective, the numbers of deaths and injuries from assaultive violence have certainly reached epidemic proportions in some American cities. Public health authorities have rightly called for increased efforts to address this urgent health problem by whatever means are determined to be both effective and societally acceptable. The descriptive epidemiology of homicide is clear on three salient points: 1) many homicide victims are killed by friends or acquaintances; 2) many homicides occur as a result of an argument, frequently over relatively trivial matters; and 3) approximately six out of 10 homicides are committed with firearms, usually handguns.[13] On the basis of these observations, several investigators have hypothesized that many homicides could be prevented if ready access to firearms were reduced, since many violent altercations occurring among family, friends, and acquaintances would then necessarily be resolved by less deadly means.[14-16]

The Detroit gun ordinance evaluated here is one approach to decreasing the accessibility of firearms. Theoretically, by reducing the number of persons carrying firearms on the streets of Detroit, some violent public altercations that might have resulted in fatal gunshot injuries would result in other nonfatal outcomes. Our time-series findings are consistent with a model in which the ordinance had a relatively small preventive effect on the incidence of homicides in Detroit. More importantly, the effect is found precisely where we predicted we would find it, based on our hypothesis that the ordinance would affect the incidence of firearm homicides and homicides occurring outside but not the incidence of other types of homicide.

We also found that the Detroit ordinance requiring mandatory sentences was almost completely unenforced. If the ordinance was never enforced, how can any preventive effect, small or otherwise, be ascribed to it? During the period from November 1986 through January 1987, media coverage of the passage of the ordinance was extensive and public debate about the virtues of the ordinance was heated. In several articles, local officials pledged to enforce the law strictly and to provide jail space as necessary. This publicity may have been responsible for the preventive effect that we found associated with the promulgation of the ordinance. The lack of enforcement—and the coincident ending of the publicity surrounding the law's passage after January 1987—may account for the relatively small nature of the observed effect.

Little previous research has been done on the effectiveness of laws that require mandatory sentences for carrying firearms illegally. To date, the most extensive studies of this issue dealt with the Massachusetts Bartley-Fox Law which required a mandatory minimum one-year sentence for carrying a firearm illegally. Investigators reported that the Bartley-Fox law reduced the number of robberies, assaults, and homicides committed with a firearm.[5,17] A more in-depth study of assaults, however, revealed that, whereas assaults with firearms decreased, assaults without firearms increased. This raises the question of whether a reduction in firearm-carrying results in a displacement rather than a reduction in interpersonal violence. In the case of the Bartley-Fox law, the apparent increase in nonfirearm assaults appears to have been due to increased reporting of more minor assaults after the law was implemented.[17] From a public health perspective, however, this issue is moot: a displacement of interpersonal violence from altercations in which firearms are used to altercations in which firearms are not used would most likely have the desired effect of preventing many homicides.

In addition to the intuitive appeal of reducing homicides in public by reducing the public carrying of firearms, ordinances such as this one in Detroit are worthy of close evaluation for several other reasons. First, the law imposes relatively few costs on individual citizens who own and use firearms for purposes such as hunting or protection. The ordinance does not prevent a person from keeping a firearm in the home. In addition, under the ordinance a firearm may be legally transferred from place to place (e.g., from the home to a firing range or out to a hunting area) as long as it "is unloaded in both barrel and magazine and is taken down, enclosed in a case, carried in the luggage compartment of the vehicle or inaccessible from the interior of the vehicle." The ordinance only prohibits carrying a concealed, loaded pistol on one's person, or any

APPENDIX

Noise Model:	Intervention Model:
Inside Homicides	
ARIMA(0,0,0) $a_t = Y_t - \Theta_0$ $\hat{\Theta}_0 = 25.95$ s.e. = .7429 Box-Ljung Q = 24 with 25 degrees of freedom	$Y_t^* = a_t + \omega_0 (I_t)$ $I_t = 1$ for months 1–84 $I_t = 0$ for months 85–96 $\hat{\omega} = 5.798$ s.e. = 2.1011
Outside Homicides	
ARIMA$(1,0,0)(1,0,0)_{12}$ $a_t = (1 - \phi_1 B)(1 - \phi_{12} B^{12}) Y_t - \Theta_0$ $\hat{\Theta}_0 = 20.83$ s.e. = 1.3496 $\hat{\phi}_1 = .3220$ s.e. = .0970 $\phi_{12} = .3529$ s.e. = .0957 Box-Ljung Q = 19 with 23 degrees of freedom	$Y_t^* = a_t + \omega_0 (I_t)$ $I_t = 0$ for months 1–84 $I_t = 1$ for months 85–96 $\hat{\omega} = 2.086$ s.e. = 2.5771
Nonfirearm Homicides	
ARIMA(0,0,0) $a_t = Y_t - \Theta_0$ $\hat{\Theta}_0 = 15.56$ s.e. = .4920 Box-Ljung Q = 15 with 25 degrees of freedom	$Y_t^* = a_t + \omega_0 (I_t)$ $I_t = 0$ for months 1–84 $I_t = 1$ for months 85–96 $\hat{\omega} = 2.524$ s.e. = 1.3918
Firearm Homicides	
ARIMA$(1,0,0)(1,0,0)_{12}$ $a_t = (1 - \phi_1 B)(1 - \phi_{12} B^{12}) Y_t - \Theta_0$ $\hat{\Theta}_0 = 31.42$ s.e. = 1.54[illegible] $\hat{\phi}_1 = .2791$ s.e. = .1016 $\phi_{12} = .2301$ s.e. = .1115 Box-Ljung Q = 19 with 23 degrees of freedom	$Y_t^* = a_t + \omega_0 (I_t)$ $I_t = 0$ for months 1–84 $I_t = 1$ for months 85–96 $\hat{\omega} = 4.109$ s.e. = 3.4885

loaded firearm in a car in such a manner that it could readily be retrieved.

Second, the notion that local governments have the right to determine who may legally carry loaded firearms in their jurisdictions appears to be relatively well accepted and not subject to much of the controversy surrounding "gun control." Indeed, the Detroit gun ordinance may be considered innovative only because of the inclusion of mandatory sentencing provisions. If such laws are generally considered to represent a reasonable function of local government, then—should scientific study demonstrate their utility in preventing homicide—ordinances like the one in Detroit might be adopted in other jurisdictions with relatively little controversy.

In contrast to the minor costs imposed by the ordinance on individual citizens, however, the general costs associated with arrest, adjudication, and mandatory incarceration of violators of the ordinance are *not* trivial. As noted in our evaluation of the enforcement of the Detroit ordinance, jail time is frequently both a scarce and expensive resource, and mandatory sentence laws, if enforced, would certainly increase the numbers of persons in jail, at least in the short term.

Given the dearth of similar research, the relatively small magnitude of the effect observed in Detroit, and the fact that the Detroit ordinance was not enforced, our findings must not be considered definitive. Reasonable caution requires that the study be replicated in a setting in which a similar ordinance is effectively and visibly enforced. Despite the theoretical and practical appeal of the Detroit gun ordinance, assessing the effectiveness of such ordinances in preventing homicide is imperative before public health officials advocate their wider adoption by other localities. In addition, the feasibility of proper, visible, and ongoing enforcement of such laws should also be assessed. Merely passing an ordinance to decrease the carrying of loaded firearms in public is clearly not adequate. Moreover, passing but not enforcing such ordinances may have unanticipated negative consequences. The inevitable ineffectiveness of an unenforced ordinance might lead policy makers to the inappropriate conclusion that such ordinances are *inherently* ineffective. If laws are passed to curtail the number of people carrying loaded firearms in public, they must be enforced. If such laws are passed *and* enforced, then the public health community should miss no opportunity to evaluate the effect of these laws on the rate of injury and death from firearms violence in public. □

Acknowledgments

We gratefully acknowledge William F. Hart, Chief of Police, Detroit Police Department; Adger Butler, PhD, Jack Samuels, PhD, and Jim Klein for their assistance in the conduct of this study. We would especially like to thank Michael Fried, Chief, Administration Division of the Wayne County Prosecutor's Office, who opened doors for us and provided many useful insights.

This paper was presented at the annual meeting of the American Society for Criminology in Reno, Nevada, November 10, 1989.

The statistical analysis presented herein was independently reviewed and confirmed by Daniel J. Schnell, PhD, Mathematical Statistician, Division of STD/HIV Prevention, Center for Prevention Services, Centers for Disease Control.

References

1. Federal Bureau of Investigation: Crime in the United States: Uniform Crime Reports for the United States. Washington, DC: FBI, annual.
2. Swickard J: Detroit Tops Big Cities in Rate of Youths Slain. Detroit Free Press Sept. 11 1988; 1A, 15A.
3. Centers for Disease Control: Impact of homicide on years of potential life lost in Michigan's black population. MMWR 1989; 38:4–11.
4. Detroit City Council: Ordinance No. 25-86, Chapter 38, Article X, Sections 1,11,58, 60 amended; Sections 9,57 repealed. Nov. 1986.
5. Rossman D, Paul F, Pierce GL, McDevitt J, Bowers W: Massachusetts' mandatory minimum sentence gun law: Enforcement, prosecution, and defense impact. Criminal Law Bull 1980; 61:150–163.
6. Beha JA: And nobody can get you out: The impact of a mandatory prison sentence for the illegal carrying of a firearm on the use of firearms and on the administration of criminal justice in Boston. Boston U Law Rev 1977; 57:96–146, 290–333.
7. US Department of Justice, National Institute of Law Enforcement and Criminal Justice: The nation's toughest drug law: Evaluating the New York experience. Final report of the Joint Committee on New Drug Law Evaluation. Washington, DC: Govt Printing Office, 1978.
8. Carlson K: Mandatory sentencing: The experience of two states: National Institute of Justice policy brief. Washington, DC: US Department of Justice, 1982.
9. Loftin C, Heumann M, McDowall D: Mandatory sentencing and firearms violence: Evaluating an alternative to gun control. Law Soc Rev 1983; 17:287–318.
10. Box GEP, Tiao GC: Intervention analysis with applications to economic and environmental problems. J Am Stat Assoc 1975; 70:70–79.
11. Box GEP, Jenkins GM: Time series analysis: Forecasting and control. San Francisco: Holden-Day, 1976.

12. US Department of Health and Human Services, US Department of Justice: Report of the Surgeon General's workshop on violence and public health. Leesburg, VA: Health Resources and Services Administration, 1986.
13. Centers for Disease Control: Homicide surveillance, 1970–1983: High-risk racial and ethnic minorities—blacks and Hispanics. Atlanta: CDC, 1985.
14. Zimring F: Is gun control likely to reduce violent killings? U Chicago Law Rev 1968; 35:721–737.
15. Cook PJ: The influence of gun availability on violent crime patterns. *In:* Tonry M, Morris N (eds): Crime and Justice: An annual review of research. Chicago: University of Chicago Press, 1983; 49–89.
16. Baker SP: Without guns, do people kill people? Am J Public Health 1985; 75:587–588.
17. Pierce GL, Bowers W: The Bartley-Fox gun law's short-term impact on crime in Boston. Ann Am Acad Politic Soc Sci 1981; 455:120–137.

Fellowship Fund at UCLA to Benefit American Indian Students

The George Blue Spruce, Jr., Fellowship Fund for American Indian Students was recently established at the University of California at Los Angeles, School of Public Health. Named in recognition of George Blue Spruce, Jr., of Phoenix, Arizona, the fellowship will provide for scholarships and loans to American Indian students preparing for careers in the field of public health. The fellowship is administered by the Health Careers Opportunity Program at the school, under direction of a committee composed of American Indian alumni from the UCLA School of Public Health.

Dr. Blue Spruce, who comes from the San Juan/Laguna Pueblo Tribes of New Mexico, was the first American Indian to graduate as a dentist in the United States. His doctorate of dental surgery is from Creighton University. Blue Spruce's career in health service includes work as a US Navy dental officer, clinical assignments on Indian reservations, assistant surgeon general in the US Public Health Service, director of the Office of Native American Programs in the US Office of Human Development, and consultant in dental health for the World Health Organization, In addition, he has chaired the Intra-Departmental Council of Indian Affairs in Washington, DC and was director of the Phoenix Area Indian Health Services for the Department of Health Services.

To date, approximately 30 American Indians have completed advanced degree programs at the UCLA SPH. The George Blue Spruce, Jr., Fellowship Fund is the third fund established at the school to support the recruitment and retention of minority students. In 1987, the Richard Chavez Scholarship Fund was established for Hispanic students, and in 1990 the Betty Smith Williams Scholarship Fund was established for African-American students.

For further information and donations to the fellowship, contact: Health Careers Opportunity Program, UCLA School of Public Health, 41-240, Los Angeles, CA 90024-1772.

Cervical Papillomavirus Infection and Cervical Dysplasia in Hispanic, Native American, and Non-Hispanic White Women in New Mexico

ABSTRACT

Background: Human papillomavirus infections of the cervix are found with varying frequencies in different populations worldwide, and have been associated with cervical cytologic abnormalities.

Methods: We studied 1,603 randomly selected Hispanic, Native American, and non-Hispanic White women in New Mexico to determine the prevalence of cervical HPV infection in these ethnic groups, and its association with Pap smear abnormalities, using a new commercial dot-blot hybridization assay.

Results: Nine percent of all women screened had evidence of cervical HPV infection (13.7% of non-Hispanic White women, 9.7% of Hispanics, and 6.6% of Native American women). Prevalence was higher in younger women ages 14–19 years than in older age groups. Over half of women with cervical HPV infection (n = 145) had normal Pap smears. The proportion of infected women increased among those with more advanced cytopathologic abnormalities; 5.6 percent with normal Pap smears had cervical HPV vs 66.7 percent with moderate-severe dysplasia.

Conclusions: Cervical HPV infection is common among New Mexico clinic attendees, varies in prevalence among the three major ethnic groups, and is strongly associated with cervical cytopathologic abnormalities. (*Am J Public Health* 1991;81:582–586)

Thomas M. Becker, MD, Cosette M. Wheeler, PhD, Nancy S. McGough, RN, Scott W. Jordan, MD, Maxine Dorin, MD, and Jill Miller, MD

Introduction

Human papillomavirus (HPV) infections of the genital tract are among the most common sexually transmitted viral infections in the United States.[1-3] Recent estimates have suggested that as many as 12 million adults in this country may be infected with genital HPV.[3] In addition to causing genital warts, HPV infections may also result in clinically inapparent lesions of the cervix. Cervical HPV infection can be diagnosed by certain morphologic features on colposcopy,[4] by cytopathologic changes seen in Papanicolaou (Pap) smears,[5] through detection of HPV antigen by immunoperoxidase stains,[6] or by identification of HPV DNA sequences in hybridization studies.[7,8] Hybridization techniques, considered more sensitive and specific than most other types of HPV tests,[9] have shown the presence of HPV genomes in 6–30 percent of women tested for cervical abnormalities from various populations worldwide.[10-15] The use of recently developed polymerase chain reaction-based HPV diagnostic tests suggests that cervical HPV prevalence may be even higher than has been determined by hybridization methods.[16,17]

New Mexico's population is comprised primarily of three major ethnic groups—Hispanics, Native Americans, and non-Hispanic Whites. In prior reports, we have shown variation in the incidence and prevalence of many infectious and chronic diseases among these ethnic groups,[18-22] including invasive and pre-invasive cervical cancer.[23] As part of an ongoing cohort study to investigate the natural history of cervical HPV infection, we studied Hispanic and non-Hispanic White women attending the University of New Mexico (UNM) gynecology clinics, and Native American women attending local Indian Health Service (IHS) hospital clinics to determine ethnic differences in the prevalence of cervical HPV infection using a newly developed hybridization test, and the relationship of cervical HPV infection to cervical cytologic abnormalities in these ethnic populations.

Methods

Study Sample

Subjects were women who attended the UNM gynecology clinics and the Albuquerque IHS hospital-affiliated clinics, June 1988–September 1989. On randomly selected days of the week, providers screened all women on whom a pelvic examination was performed for any reason. This sampling scheme was consistent among clinics and sites and all five clinic days (Monday–Friday) were equally likely to be selected. Patients who underwent a pelvic examination for any reason were included in this survey. The UNM clinic attendees were comprised primarily of Hispanic and non-Hispanic White women; few women of other ethnic or racial groups attend these clinics. At the UNM clinics, Hispanic ethnicity is either

---

Address reprint requests to Thomas M. Becker, MD, Assistant Professor, Department of Medicine, Cancer Center, School of Medicine, University of New Mexico, Albuquerque, NM 87131. Dr. Wheeler is Research Assistant Professor, Department of Cell Biology; Ms. McGough is Research Nurse, Department of OB/GYN; Dr. Jordan is Professor, Department of Pathology; Dr. Dorin is Assistant Professor, Department of OB/GYN; Dr. Miller is Clinical Adjunct Professor, Department of Family Medicine; all at UNM School of Medicine. This paper, submitted to the Journal April 17, 1990, was revised and accepted for publication January 8, 1991.

determined by self-identification at the time of clinic enrollment or is assigned by clinic staff on the basis of Spanish surname. To validate the UNM clinics' designation of Hispanic and non-Hispanic White ethnicity, we interviewed a 15 percent sample of the UNM clinic group studied. We found 91 percent concordance between the self-reporting of Hispanic or non-Hispanic White ethnicity compared to the combined self-identified/clinic staff assignment of ethnic status. The Indian Health Service hospital clinics serve primarily Pueblo and Navajo women. No attempt was made to validate Native American ethnicity. We did not include in our report those Black women who were screened at the UNM clinics for cervical HPV (n = 7). Informed consent was required at three of the four clinics affiliated with the UNM gynecology department. Only two refusals to participate in the screening survey were recorded among all clinic patients; both of these patients were of Hispanic ethnicity.

UNM clinics serve primarily women who are economically disadvantaged. The IHS-affiliated clinics provide services free of charge to eligible Native American patients. Nonetheless, Native Americans in New Mexico are an economically disadvantaged group: approximately 40 percent of the state's Native American population lived below the poverty level in 1980.[24]

Cytopathologic Diagnoses

Pap smears from the UNM clinics were all read by the same cytopathologist (SWJ); cytologic smears from the IHS hospital were initially read by pathologists from Path Labs, El Paso, Texas. We found 98 percent concordance between these two laboratories for diagnoses of a sample of normal cervical smears (n = 59). The UNM cytopathologist (SWJ) reviewed all abnormal Pap smears which were initially reported by Path Labs for the IHS clinic attendees. To maintain consistency in cytopathologic diagnoses for this report, we used the UNM cytopathologist's diagnoses for abnormal Pap smears in our analysis. Cytopathologic changes associated with HPV infection (such as koilocytosis) were coded as atypias for this report. All diagnoses corresponded to the World Health Organization's criteria for cervical cellular abnormalities.[25] During the period of this survey, the newly developed Bethesda Classification System for cervical cytology had not been implemented in either pathology laboratory.[26]

HPV Testing

We used a recently developed dot hybridization screening test, ViraPap® (Life Technologies, Inc, Gaithersburg, MD 20857) to test for cervical HPV infection. This test is designed to identify genital HPV types 6, 11, 16, 18, 31, 33, and 35—the types most commonly associated with genital tract infection. The test includes sampling the endo- and exocervix with a dacron-tipped plastic swab for exfoliated cells. HPV samples were collected after the Pap smears were collected. Dacron swabs were placed immediately in tubes of ViraPap™ collection media. The tubes were frozen at −20°C until testing in batch at the HPV laboratory, University of New Mexico School of Medicine. All positive HPV ViraPap tests underwent typing using the ViraType® test (Life Technologies, Gaithersburg, MD 20857). This dot hybridization test is able to separate HPV genome into the three categories, 6/11, 16/18, and 31/33/35, but does not determine which of the virus types is present within a given category. All borderline test results by ViraPap were further tested by ViraType and considered negative only if they were negative by the ViraType test.

The HPV laboratory independently validated the sensitivity and specificity of ViraPap and ViraType using Southern Blot hybridization techniques as the "gold standard".[16,17] For 147 specimens, the sensitivity of the ViraPap assay was 100 percent and specificity 96 percent. For 63 specimens, the sensitivity of the ViraType assay was 100 percent and specificity 91 percent.

Data Analysis

Data were analyzed using standard packages from SAS.[27] We examined the age-adjusted prevalence of cervical HPV infection for Hispanic, Native American, and non-Hispanic White women between the ages of 15 to 44 years using the combined population distribution for these study patients in this age range as our reference population. We age-adjusted these figures using the direct method of standardization.[28]

Results

We studied 535 Hispanic, 322 non-Hispanic White, and 746 Native American women, a total of 1,603 women. The mean ages of the participants were 25.0 years for the Hispanic women (range 15–43 years), 25.5 years for non-Hispanic Whites (range 14–50 years), and 29.5 years for Native American women (range 14–78 years). The prevalence of cervical HPV infection in this sample was higher for non-Hispanic White women (13.7 percent) than for Hispanic (9.7 percent), or Native American women (6.6 percent). The age-adjusted prevalence of cervical HPV infection was 13.1, 9.3, and 7.7 percent for non-Hispanic White, Hispanic, and Native American women, respectively, between the ages of 15 to 44 years. Among all women with cervical HPV infection, Native American women had a higher proportion of HPV 6/11 compared to non-Indian women (Table 1).

Younger women were more likely to have cervical HPV than were women in older age groups (Table 2). Comparable age-related decreases were observed in each of the three ethnic populations which we studied (data not shown). For all women combined, 6.7 percent of patients screened had slight, moderate, or severe dysplasia. American Indian women had a higher proportion of Pap smears showing atypias, compared to Hispanic and non-

TABLE 1—Cervical HPV Test Results by Ethnic Group and HPV Type, Cervical HPV Infection Survey (n = 1603)

		HPV Types			
Ethnic Group	HPV Positive No. (%)	6/11 No. (%)	16/18 No. (%)	31/33/35 No. (%)	Mixed[b] No. (%)
Hispanic n = 535	52 (9.7)	3 (5.8)[a]	32 (61.5)	16 (30.8)	1 (1.9)
non-Hispanic White n = 322	44 (13.7)	1 (2.3)	15 (34.1)	26 (59.1)	2 (4.5)
American Indian n = 746	49 (6.6)	6 (12.2)	18 (36.7)	23 (46.9)	2 (4.2)

a) Percent of all HPV-positive specimens within that ethnic population.

b) Mixed virus types include combinations of 6/11 and 31/33/35, (n = 1), and 16/18 and 31/33/35 (n = 2), and 6/11 and 16/18 (n = 2).

TABLE 2—Cervical HPV Test Results by Age Group for all Ethnic Groups Combined, Cervical HPV Infection Survey (n = 1603)

	Age Group (years)						
HPV Results	14–19 No. (%)	20–29 No. (%)	30–39 No. (%)	40–49 No. (%)	50–59 No. (%)	60+ No. (%)	Unknown No. (%)
Negative	159 (86.9)	811 (89.7)	378 (93.8)	69 (98.6)	20 (100)	13 (100)	6 (75.0)
Positive	24 (13.1)	93 (10.3)	25 (6.2)	1 (1.4)	0	0	2 (25.0)
Total	183	904	403	70	20	13	8

TABLE 3—Cytopathologic Diagnoses for Hispanic, Non-Hispanic White, and American Indian Participants, Cervical HPV Infection Survey

	Negative No. (%)	Atypia No. (%)	Slight Dysplasia No. (%)	Moderate-Severe Dysplasia No. (%)
Hispanic n = 535	499 (93.3)	4 (.8)	25 (4.7)	7 (1.3)
non-Hispanic White n = 322	284 (88.2)	3 (.9)	27 (8.4)	8 (2.5)
American Indian n = 746	664 (89.0)	45 (6.0)	25 (3.4)	12 (1.6)
Total n = 1603	1447 (90.3)	52 (3.2)	77 (4.8)	27 (1.7)

Hispanic white women (Table 3). Table 4 shows that a large proportion of the 145 women with evidence of cervical HPV infection had normal Pap smears (55.9 percent), but that the prevalence of HPV increased sharply with the degree of dysplasia found in the Pap smear.

Discussion

Our data show ethnic differences in the prevalence of cervical HPV infection, as well as ethnic differences in types of HPV present on the cervix at the time of the screening examination, and substantiate prior published observations that HPV is more commonly found in more advanced cervical lesions.[3,12] Unlike most prior reports, however, our survey showed that HPV types 31, 33, and 35 were often present in association with cytologic abnormalities.

The prevalence of cervical HPV infection in different populations worldwide varies substantially, depending on the diagnostic test employed for HPV detection. Using cytologic criteria, Meisels and Morin[5] reported that 1.7 percent of 234,715 women who participated in a screening program in Quebec, Canada, between 1975–1979 had cytologic evidence of cervical HPV infection. In other cytologic surveys, prevalence estimates ranged from 0.9 percent to 16 percent.[1,29-33] Among these reported series, the prevalence has been higher in sexually transmitted disease clinics[29,32,33] than in other clinic populations. Research which has employed DNA hybridization techniques found evidence of cervical HPV DNA to vary from 6 to 28 percent of women tested,[10-15] with several studies reporting a prevalence of about 10 percent.[11-15,29,33] Using Southern blot hybridization for evidence of HPV 6, 11, 16, or 18 DNA, deVilliers, *et al*,[12] found the highest prevalence of HPV DNA was from cervical specimens from women less than 25 years of age (14 percent), and the lowest prevalence among women age 56 or older (4 percent). Similarly, Kiviat, *et al*, reported a higher prevalence of cervical HPV DNA in women 16–24 years old who attended a sexually transmitted disease clinic or a student health clinic in Seattle, Washington compared with older age groups of women at both clinics.[33]

HPV types 16, 18, and 31 have been implicated as having a more aggressive role in malignant transformation of cervical cells.[34-38] Our screening data cannot address the issue of malignant transformation; however, if published observations on the malignant potential of HPV 16, 18, and 31 are accurate,[11,36] many of the clinic attendees we studied in New Mexico are at high risk for development of cervical cancer. Nonetheless, current epidemiologic evidence is too weak to accurately assess the relationship of specific HPV types in genital cancer etiology: carefully designed prospective studies are necessary which consider other potential risk factors in the development of disease. Ethical considerations, however, cannot allow clinicians to follow patients to the end-point of cervical cancer without earlier intervention; thus, the appropriate studies to determine the relationship between HPV infection and cervical cancer will probably never be carried out. Population-based screening for HPV (by type), together with examination of cervical cancer specimens for type-specific HPV genome from cases which occur in those same populations, may shed additional light on the relationship of specific HPV types in cervical cancer.

Although several case-control studies and one cohort study have indicated that the presence of HPV in cervical tissue is a strong risk factor for cervical dysplasia

TABLE 4—Cytopathologic and HPV Test Results by HPV Type, all Ethnic Groups Combined, Cervical HPV Infection Survey (n = 1603)

		HPV type			
Pap Smear Diagnosis	HPV (+) No. (%)	6/11[a] No. (%)	16/18 No. (%)	31/33/35 No. (%)	Mixed No. (%)
Negative n = 1447	81 (5.6)	8 (9.9)	34 (42.0)	37 (45.7)	2 (2.5)
Atypia n = 52	11 (21.2)	0 (0)	7 (63.6)	4 (36.4)	0 (0)
Slight n = 77	35 (45.5)	2 (5.7)	13 (37.1)	17 (48.6)	3 (8.6)
Mod-Severe n = 27	18 (66.7)	0 (0)	11 (61.1)	7 (38.9)	0 (0)

a) % of all specimens which showed evidence of HPV within the Pap smear diagnostic category.

or cervical cancer,[2,10,12,14,38,39] data on genital cancer incidence in New Mexico's Hispanics, Native Americans, and non-Hispanic Whites when compared to our survey findings appear to provide evidence that conflicts with these studies. The highest cervical cancer rates in New Mexico occur among Native American and Hispanic women, with rates for non-Hispanic White women which are comparable to those rates for whites nationwide[23]—the exact opposite of our survey of HPV prevalence. Although such ecological correlations are weak and do not consider numerous confounding factors in the infection-disease relationship, they suggest that we examine the epidemiology of cervical disease as related to HPV infection and to other relevant behavioral, infectious, and dietary factors more closely.

Our study has limitations that must be addressed. The HPV laboratory test we used, while demonstrating high sensitivity and specificity compared to Southern Blot hybridization for certain HPV types, is unable to determine the presence of genital HPV types other than those included in the test kit. The New Mexico clinic populations may thus contain an even greater proportion of women infected with other genital HPV types which have been identified in other populations.[40–42] Furthermore, implementation of highly sensitive polymerase chain reaction-based HPV diagnostic testing in other populations suggests that cervical HPV prevalence is considerably higher than can be determined by using hybridization methods.[16,17] Thus, it is likely that cervical HPV prevalence in our clinic populations may be higher as assessed through polymerase chain reaction techniques than we report using the commercial dot-blot assay. In addition, we have examined single specimens for the presence of cervical HPV infection; serial sampling of the same patients may result in the determination of an even greater number of women with cervical HPV infection. Poor technique in the collection of Pap smears or HPV samples could also influence our results; however, all clinicians who collected these specimens were instructed in proper sample collection techniques. Despite these potential sources of bias in our study, we have demonstrated that cervical HPV infection is common among university-affiliated and Indian Health Service hospital-affiliated clinic attendees in New Mexico; prospective studies are ongoing to evaluate the relationship of cervical HPV infection to the development of dysplastic lesions in these populations. ☐

Acknowledgments

This research was supported by a contract from the Indian Health Service, Contract No. 249-88-0013. We wish to thank Drs. William Reeves, Joseph Icenogle, Kay Stone, and Carlos Lopez of the Centers for Disease Control for advice and assistance, and Dorothy Fordyce for secretarial assistance. We are grateful to the staff at the Women's Health Care Clinic and the Maternal and Infant Care clinics, and to the staff at the Albuquerque Indian Health Service Hospital for their help in specimen collection. In particular, Diane Sampson, Sandi Jensen, and Marianne Wheeler assisted with specimen collection. We also appreciate the advice of Drs. Andrew Kramer, Del Endres, Charles North, and Eric Henley. Lynne Fullerton and Corinne Peek assisted with data collection. Dr. Richard Juel of Path Labs, El Paso, Texas, kindly provided cytologic smears for review.

References

1. Becker TM, Stone KM, Alexander ER: Genital human papillomavirus infection: A growing concern. Obstet Gynecol Clin N Am 1987; 14:389–396.
2. Chuang T-Y, Perry HO, Kurland LT, *et al:* Condyloma acuminatum in Rochester, Minnesota, 1950–1978, Parts I and II. Arch Dermatol 1984; 120:469–483.
3. Koutsky LA, Galloway DA, Holmes KK: Epidemiology of genital human papillomavirus infection. Epidemiol Rev 1988; 10:122–163.
4. Coppleson M: Colposcopic features of papillomaviral infection and premalignancy in the female lower genital tract. Obstet Gynecol Clin N Am 1987; 14:471–494.
5. Meisels A, Morin C: Human papillomavirus and cancer of the uterine cervix. Gynecol Oncol 1981; 12:S111–S123.
6. Gupta JW, Gupta PK, Shah KV, *et al:* Distribution of human papillomavirus antigen in cervicovaginal smears and cervical tissues. Int J Gynecol Pathol 1983; 2:160–170.
7. Gissman L, Zur Hausen H: Human papillomavirus: Physical mapping and genetic heterogeneity. Proc Natl Acad Sci USA 1976; 73:1310–1313.
8. Lorincz AT: Detection of human papillomavirus infection by nucleic acid hybridization. Obstet Gynecol Clin N Am 1987; 14:451–469.
9. Schneider A, Meinhardt G, deVilliers E-M, *et al:* Sensitivity of the cytologic diagnosis of cervical condyloma in comparison with HPV DNA hybridization studies. Diagn Cytopathol 1987; 3:250–255.
10. Pratili MA, LeDoussal V, Harvey P, *et al:* Recherche de papillomavirus humains dans des cellulules epitheliales du col uterin: Frequence des types 16 et 18. J Gynecol Obstet Biol Reprod (Paris) 1986; 15:45–50.
11. Lorincz AT, Temple GF, Patterson JA, *et al:* Correlation of cellular atypia and human papillomavirus deoxyribonucleic acid sequences in exfoliated cells of the uterine cervix. Obstet Gynecol 1986;68:508–512.
12. deVilliers E-M, Wagner D, Schneider A, *et al:* Human papillomavirus infections in women with and without abnormal cervical cytology. Lancet 1987; 2:703–706.
13. Toon PG, Arrand JR, Wilson LP, *et al:* Human papillomavirus infection of the uterine cervix of women without cytological signs of neoplasia. Br Med J 1986; 293:1261–1264.
14. Schneider A, Hotz M, Gissmann L: Increased prevalence of human papillomavirus in the lower genital tract of pregnant women. Int J Cancer 1987; 40:198–201.
15. Fife KH, Rogers RE, Zwickl BW: Symptomatic and asymptomatic cervical infections with human papillomavirus during pregnancy. J Infect Dis 1987; 156:904–911.
16. Pao CC, Lin C, Maa JS, *et al:* Detection of human Papillomavirus in cervicovaginal cells using polymerase chain reaction. J Infect Dis 1990; 161:113–115.
17. Manos MM, Lee K, Greer C, *et al:* Looking for human Papillomavirus 16 by PCR. Lancet 1990; 1:734–735.
18. Becker TM, Wiggins CL, Peek C, *et al:* Mortality from infectious diseases among New Mexico's American Indians, Hispanic Whites, and other Whites, 1958–1987. Am J Public Health 1990; 80:320–323.
19. Becker TM, Madger L, Harrison HR, *et al:* The epidemiology of infection with the human herpes viruses in Navajo children. Am J Epidemiol 1988; 127:1071–1078.
20. Becker TM, Wiggins CL, Key CR, Samet JM: Ischemic heart disease mortality in Hispanics, American Indians, and non-Hispanic Whites in New Mexico, 1958–82. Circulation 1988; 78:302–309.
21. Samet JM, Wiggins CL, Key CR, Becker TM: Mortality from lung cancer and chronic obstructive lung disease in New Mexico, 1958–1982. Am J Public Health 1988; 78:1182–1186.
22. Wiggins CL, Becker TM, Key CR, *et al:* Stomach cancer among New Mexico's American Indians, Hispanic Whites, and non-Hispanic Whites. Cancer Res 1989; 49:1595–1599.
23. Key CR: Cancer incidence and mortality in New Mexico, 1973–1977. National Cancer Institute Monograph No. 57. Washington, DC: Govt Print Office, 1981.
24. US Bureau of the Census: Census of the Population: 1980. General population characteristics. Final Report PC80-1-B33 New Mexico. Washington, DC: Govt Print Office, 1981.
25. Riotton G, Christopherson WM, Lunt R: Cytology of the female genital tract. *In:* International Histologic Classification of Tumors, No. 8. Geneva: World Health Organization, 1973.
26. National Cancer Institute: The 1988 Bethesda system for reporting cervical/vaginal cytological diagnoses. J Am Med Assoc 1989;262:931–934.
27. SAS Institute Inc: SAS User's Guide: Statistics. Version 5 edition. Cary, NC: SAS Institute, Inc, 1985.
28. Colton T: Statistics in Medicine. Boston: Little, Brown and Company, 1974.
29. Stone KM: Epidemiologic aspects of genital HPV infection. Clinical Obstet Gynecol 1989; 32:112–116.
30. Drake M, Mitchell H, Medley G: Human papillomavirus infection of the cervix in Victoria, 1982–1985. Med J Aust 1987; 147:57–59.

31. Zaninetti P, Franceschi S, Baccolo M, *et al:* Characteristics of women under 20 with cervical intraepithelial neoplasia. Int J Epidemiol 1986; 15:477–482.
32. Armstrong BK, Allen OV, Brennan BA, *et al:* Time trends in the prevalence of cervical cytological abnormality in women attending a sexually transmitted disease clinic and their relationship to trends in sexual activity and specific infections. Br J Cancer 1986; 54:669–675.
33. Kiviat N, Koutsky LA, Paavonen JA, *et al:* Prevalence of genital papillomavirus infection among women attending a college student health clinic or a sexually transmitted disease clinic. J Infect Dis 1989; 159:293–302.
34. Schneider A, Kraus H, Schuhmann R, *et al:* Papillomavirus infection of the lower genital tract: Detection of viral DNA in gynecological swabs. Int J Cancer 1985; 35:443–448.
35. Reid R, Greenberg M, Jenson AD, *et al:* Sexually transmitted papillomavirus infections. I. The anatomic distribution and pathologic grade of neoplastic lesions associated with different viral types. Am J Obstet Gynecol 1987; 156:212–222.
36. Lorincz AT, Temple GF, Kurman RJ, *et al:* Oncogenic association of specific human papillomavirus types with cervical neoplasia. JNCI 1987; 79:671–677.
37. Barnes W, Delgado G, Kurman R, *et al:* Possible prognostic significance of human papillomavirus type in cervical cancer. Gynecol Oncol 1988; 29:267–273.
38. Reeves WC, Caussy D, Brinton LA, *et al:* Case-control study of human papillomaviruses and cervical cancer in Latin America. Int J Cancer 1987; 40:450–454.
39. Meanwell CA, Cox MF, Blackledge G, *et al:* HPV 16 DNA in normal and malignant cervical epithelium: Implications for the aetiology and behaviour of cervical neoplasia. Lancet 1987; 1:703–707.
40. Lorincz AT, Quinn AP, Goldborough MD, *et al:* Cloning and partial DNA sequencing of two new human papillomavirus types associated with condylomas and low-grade cervical neoplasia. J Virol 1989; 63:2829–2834.
41. Shimoda K, Lorincz AT, Temple GF, *et al:* Human papillomavirus Type 52: A new virus associated with cervical neoplasia. J Gen Virol 1988;69:2925–2928.
42. Nuovo GJ, Crum CP, deVilliers E-M, *et al:* Isolation of a novel human papillomavirus (Type 51) from a cervical condyloma. J Virol 1988;62:1452–1455.

New Report from Bureau of Health Professions

The Health Resources and Services Administration's Bureau of Health Professions (BHPr) recently announced publication of the summary report of its *Workshop on Modeling and Forecasting for the 1990s* (DHHS Pub. No. HRS-P-OD-90-5). This invitational workshop was sponsored by BHPr's Office of Data Analysis and Management. It focused on the desirability of utilizing a common framework of economic and health sector assumptions for all Bureau modeling and forecasting efforts.

The report summarizes the formal presentations and structured discussions of the more than 50 health services researchers from federal and state government, universities, and private research firms at the January 1990 workshop. Appendices include the workshop keynote address, agenda, schematics of BHPr models, selected references, and participant feedback.

Single copies of the report may be obtained by writing the Office of Data Analysis and Management, Howard V. Stambler, Director, BHPr, Rm. 8-47 Parklawn Bldg., 5600 Fishers Lane, Rockville, MD 20857; (301) 443-6936.

Urolithiasis in Tennessee: An Occupational Window into a Regional Problem

Michael J. Thun, MD, MS, and Susan Schober, PhD

ABSTRACT

Background: Urinary tract stones (stones) are believed to be unusually common in the southeastern United States but neither the incidence of nor the risk factors for stones are known.

Methods: In three well-defined occupational populations in eastern Tennessee, we assessed the prevalence, incidence, and cumulative incidence of stones and measured biochemical risk factors for lithogenesis.

Results: The age-adjusted prevalence of stones was 18.5 percent in Tennessee compared to 7.7 percent among White males in US NHANES (prevalence ratio 2.4, 95% CI 1.7, 3.3). The cumulative incidence (risk) was 27.8 percent by age 65, higher than in any other reported population. Risk factors were age, a family history, and urinary saturation with calcium-oxalate (COAX). Persons with a positive family history and the highest measured CAOX index had a predicted lifetime risk of 88.8 percent. Biochemical factors affecting lithogenesis were hypercalcuria, hyperoxaluria, and low urine volume.

Conclusion: Future research should characterize the geographic boundaries of a southeastern "stonebelt" and explore genetic and dietary hypotheses that might explain it. (*Am J Public Health* 1991;81:587–591)

Introduction

Kidney stones, an important source of morbidity, suffering, and medical expense, have received little epidemiologic attention. Three studies suggest an endemic area of stones in the Southeastern United States, but these rely upon hospital discharge data rather than incidence.[1–3] The absence of population-based incidence data in the US and the paucity of etiologic studies impede the identification of potentially modifiable risk factors such as diet and climate.[4,5]

The present study began as an investigation of reported urinary tract disorders among uranium workers in eastern Tennessee. Although no occupational association was found,[6] the study provided an opportunity to assess stone occurrence and risk factors for lithogenesis in a high-risk population. Our data support the concept of a regional "stone belt" and suggest clues as to its etiology.

Methods

Subjects

The uranium plant processed depleted and natural uranium and thorium from 1957 to 1970, and enriched uranium after 1970. Workers eligible for the study represent a gradient of exposure to unenriched uranium, compounds for which nephrotoxicity rather than radiation limits exposure.[7–9] The target population included: 1) highly exposed active, retired, and disabled production workers with 20 years employment before 1986; 2) a low exposure group of senior office and laboratory workers; 3) minimally exposed guards, ages 40–65, employed recently at the uranium plant; and 4) unexposed dairy workers from a nearby town. The dairy workers belong to the same trade union and local community. They produce tinned evaporated milk but have no special access to discounted milk or cheese that might affect stone risk.

Initially, a pilot questionnaire was administered to senior uranium workers and to guards. Later, a more extensive questionnaire and medical study were offered to the entire target population. Both questionnaires asked identical core questions about age and stones:

- "Have you ever had a kidney stone, including gravel in your urine?"
- "How many times have you had this?"
- "When did you first have this?"
- "Where were you treated?"

Workers interviewed in person were asked to sign a medical release form. The medical questionnaire also asked about a family history of stones, treatment for urinary tract infections, length of residence in the area, diabetes, hypertension, urinary instrumentation, prostatic disease, analgesics, smoking, "moonshine," and occupational exposure to heavy metals and solvents.

Data Collection

This activity included height, weight, seated blood pressure, serum for total calcium, phosphorus, uric acid and creatinine, sterile urine for quantitative urine culture and microscopy, and urine samples for chemistry, beta-2-microglobulin,

---

From the National Institute for Occupational Safety and Health, Cincinnati, Ohio. Address reprint requests to Michael J. Thun, MD, MS, Department of Epidemiology and Statistics, American Cancer Society, 1599 Clifton Road, NE, Atlanta, GA 30329. Dr. Schober is currently with the National Institute on Drug Abuse, Rockville, MD. This paper, submitted to the Journal March 29, 1990, was revised and accepted for publication October 19, 1990.

TABLE 1—Participation in the Questionnaire and Medical Study

		Participation	
Employment Category	Number Eligible	Core Questionnaire N (%)	Full Medical Study (%)*
Uranium Exposed			
Active			
Hourly	58	58 (100)	50 (89)
Salaried	36	36 (100)	19 (59)
Retired			
Hourly	6	5 (83)	2 (40)
Salaried	10	10 (100)	2 (20)
Disabled	6	6 (100)	3 (50)
Referents			
Guards	43	43 (100)	10 (24)
Dairy Workers	51	50 (98)	37 (79)
Total	210	208 (99)	123 (62)

*Participation in medical study based upon the number (199 total) of workers present locally when testing performed.

retinol binding protein, and enzymes.[6] Urine was collected over 24 hours to measure the "stone risk profile," including eight promoters of lithogenesis (calcium, oxalate, sodium, uric acid, sulfate, phosphorus, and high or low pH), and three inhibitors (citrate, magnesium, and urine volume).[10,11] During collection, participants maintained their usual diet, fluid intake, and activity. Urine pH was measured immediately upon return of the sample; volume was determined by dilution of a known quantity of lithium. Aliquots were shipped in dry ice for analysis.[6]

Supersaturation with CAOX, dicalcium phosphate, monosodium urate, and urate was computed from measured values using a computer algorithm that considers the complex physiochemical interaction between multiple factors.[12] Supersaturation scores are expressed relative to the group mean of 176 persons without stones.[12]

Statistical Analyses

Age-specific prevalence reflects the number of workers ever having had a stone divided by the number in the corresponding age bracket, based upon age at interview. Age-adjusted prevalence was compared to White males in NHANES II[13] using maximum likelihood estimation.[14] Age-adjusted incidence reflects the age at first stone. Person years at risk (PYAR) were computed using life-table methods. Workers contribute PYAR from birth until they develop a stone or complete the interview, whichever is first. Age-adjusted incidence in Tennessee was compared to published data from the Mayo clinic,[5] using maximum likelihood estimation;[14] 95% confidence intervals used the test-based method.[14] Cumulative incidence (based on age at first stone) used the Kaplan-Meier method.[15] Factors associated with stones were identified using Student's t-test (for continuous variables) and Chi-square (for proportions). Associations were further tested using unconditional logistic regression. In this analysis, cases are persons ever reporting stones. Terms considered in the regression are age and all factors in Table 4 associated with stones at $p < 0.2$. The effect of each risk factor was assessed separately and in the presence of others. Age is retained in the final model because of the known association of stones and age. Other factors are retained if statistically significant ($p < 0.05$) or if inclusion of the factor substantially influences the coefficient for other terms. Second degree interaction was tested using stratified analyses; interaction terms are not included in the logistic model because none of these substantially modify the main effect terms.

Results

Table 1 shows participation rates by employment category. Overall, 208 eligible workers (99 percent) completed the core questionnaire. Of 199 present locally during the medical testing, 136 (68 percent) completed the medical questionnaire and 123 (62 percent) provided blood and 24-hour urine samples (full medical study). Participation was highest among active hourly workers released from duty for testing. Guards, retired, and salaried workers were either off-site, not released from duty, or had lower commitment to the study. All participants were male, 206 were White, with an average age of 52.1 and range of 38–72 years.

Medical Record Review

Forty of 208 workers reported stones (crude prevalence 19.2 percent). Of these, 37 were treated by civilian physicians, 30 (all those interviewed in person) provided medical release forms, 28 received records from the treating facility, and 25 had a stone confirmed. Stones could not be confirmed when the treating physician had retired or relocated. Confirmed stones accounted for 34 hospitalizations, 42 abdominal X-rays, five surgical extractions, and five lithotripsies. Chemical analyses of stones were recorded on 13 persons with recurrent stones; calcium oxalate was present in 12 (92 percent). Nineteen persons reported multiple stones; five of these had from five to 20, and one had 32.

Prevalence

Table 2 presents the prevalence of stones by age and occupational group. At all ages, the Tennessee workers reported more frequent stones than did White males in NHANES.[13] The age-adjusted prevalence in the Tennessee workers was 18.5 percent vs 7.7 percent in NHANES (prevalence ratio 2.4, 95% CI = 1.7, 3.3). Stone prevalence was similar in all occupational groups except guards. Prevalence in guards was 7 percent, significantly lower than that of other workers ($p = 0.02$) and similar to NHANES.[13]

Incidence and Cumulative Incidence

The age-adjusted incidence of first stones was 4.21 per 1,000 person-years, compared to 1.03 per 1,000 in Minnesota males[5] (RR = 4.1, 95% CI = 2.9, 6.0). The cumulative incidence (risk) of stone by age 60 was 23.7 percent, and by age 65 was 27.8 percent.

Characteristics Associated with Stones

Table 3 shows selected demographic and clinical attributes of persons with and without stones. Stone-formers in our study weighed more, used moonshine liquor less, reported a more frequent family history of stones and of male relatives treated for urinary infections, and were more likely to be treated for such infections themselves. Laboratory findings associated with stones are a significantly higher serum and urine calcium, and urinary supersaturation with CAOX and brushite. Findings not associated with stones in our study but of interest in the literature are serum and urine uric acid,[16] current cigarette smoking,* and analgesic use.[17]

Table 4 presents the results of unconditional logistic regression. The two factors most strongly associated with stones were the index for CAOX supersaturation in a current urine sample, and a family history of stones. The logistic model (Table 4) categorizes the CAOX index into tertiles. In Figure 1, the CAOX index is presented as a continuous variable. Figure 1 illustrates the probability of stones, as predicted by the model, in persons with and without a family history over the range of the CAOX observed in our study. Males with a family history and the highest measured CAOX index have a predicted lifetime probability of stones of 88.8 percent. Using multiple linear regression, we determined that the most important determinants of the CAOX were high urinary calcium and oxalate and low urine volume. Together, these terms explained 91.8 percent of the variance, with the individual contribution of calcium, urine volume, and oxalate being 49.5 percent, 34.1 percent, and 8.2 percent, respectively. Six other stone risk factors used to compute the CAOX index were statistically significant at the $p < 0.001$ level, but together these added only 6.7 percent to the explained variance.

TABLE 2—Age-Specific Prevalence of Stones: Tennessee Workers vs US

	Number (Percent) with Stones			
	Tennessee (N = 208)		US NHANES[18] (N = 4331)	
Age (years)	N	(%)	N	(%)
30–39	2	(20.0)	46	(5.0)
40–49	11	(13.3)	57	(7.4)
50–59	14	(21.2)	52	(7.9)
60–69	11	(23.9)	163	(9.2)
Age-adjusted	40	(18.5)	326	(7.7)

Occupation-Specific Prevalence of Stones: Tennessee Workers

Occupation	Potential Uranium Exposure	Workers N	Prevalence Stones N	(%)
Uranium workers				
Production	(High)	95	19	(20)
Office	(Low)	20	5	(25)
Guards	(Low)	43	3	(7)
Dairy workers	(None)	50	13	(26)
Total		208	40	(19)

Discussion

Our principal finding is an increased risk of stones among males in eastern Tennessee, consistent with previous reports of a regional "stone belt."[1-3] At highest risk are persons with a positive family history whose urine is supersaturated with CAOX, a major ingredient of most stones.[4] Provided that our findings reflect regional occurrence, they suggest that stones are an important public health problem in the southeast, and provide some clues about etiology.

The lifetime risk of stones among the Tennessee workers is higher than in any other reported population. Risk by age 60 is 24 percent in our study, compared to 5 percent in Minnesota,[5] 7 percent in northern California,[4] and [illegible] percent in Uppsala, Sweden.[18] These regional differences are larger than can be explained by differences in the methods for estimating risk. The Swedish data, for example,[18] are based upon a prevalence survey of renal colic in 1781 men, age 60. The US NHANES survey,[13] using similar methods but inquiring about stones instead of renal colic, show a lifetime prevalence of 9.2 percent in White males ages 60–69. The lower risks observed in the California and Minnesota studies are based upon incidence rates of physician visits.[4,5] Incidence is converted to risk using the formula [$\text{risk} = 1 - e^{(-\text{Incidence} \times \text{Time})}$].

Several findings suggest that the risk of stones in our study is regional rather than occupational. First, a family history of stones affecting first degree relatives who have never worked at either plant was common in both stone-formers (52 percent) and those without stones (21.5 percent). This can be compared to Sweden, where 29.4 percent of stone-formers and 15.3 percent of nonstone-formers report a family history.[18] Second, the occurrence of stones in the uranium workers bears no relation to the intensity or duration of occupational exposure. Stones are slightly less common in workers with direct exposure to uranium and thorium than in unexposed workers. Third, stone occurrence is similar in the uranium and dairy workers. If stone occurrence were due to occupation it would be unlikely that two such different occupational settings would cause a nearly identical effect.

The geographic boundaries of the so-called "stone-belt" are poorly defined. Four southeastern states (North Carolina, South Carolina, Virginia, and Tennessee) rank high in terms of hospitalizations for stones as a proportion of all hospital admissions.[1] There are limitations to these hospital discharge data, however,[4,5] and no studies have collected population-based data on incidence in the southeast US. Such data would be extremely useful in mapping the occurrence of stones, defining high-risk areas, and identifying preventable risk factors.

No cause has yet been identified for increased stone-formation in the southeast. Various hypotheses include water softness,[19] sunlight and heat,[20] dietary consumption of meat,[16] and carbonated beverages.[21] All of these hypotheses are based upon ecological evidence and have not been tested in studies assessing both stone occurrence and the exposure of individuals to the proposed risk factors. Our study is the first to measure biochemical risk factors in urine in a high-risk population; as such it helps to narrow the range of hypotheses to be pursued in future research. The single most important factor contributing to CAOX supersaturation in our study was increased urinary calcium.

Twenty-five (20.7 percent) of participants in the medical study had clinical hypercalcuria (>300 mg per 24 hours).[16] We did not obtain a dietary history and cannot assess whether the increased uri-

---

*Fitzsimmons SC: Self-reported kidney stone disease among Black and White adults: Prevalence and correlates in NHANES 11 (1976–1980). Presentation at 116th annual meeting of the American Public Health Association, Nov. 15, 1988, Boston.

TABLE 3—Demographic and Clinical Characteristics of Persons with and without Stones

Characteristic	Stones			No Stones			p value⁺
	Mean	(SD)	Range	Mean	(SD)	Range	
Age years*	53.5	(8.5)	38–71	51.7	(7.9)	38–72	—
Weight kg.**	92.8	(15.5)	69–135	85.4	(13.0)	56–120	(0.01)
% Born locally**	86.2			85.2			—
% Away ≥ 20 years**	9.7			19.4			(0.2)
% Ever use moonshine liquor**	25.9			61.5			(0.001)
% Family history of stones**	51.7			21.5			(0.001)
% Family history of infection**	20.7			5.7			(0.02)
% Personal history of infection**	41.1			21.5			(0.009)
% Smoking, current**	34.5			35.5			—
% Phenacetin usage**	0.0			7.2			—
Calcium, serum (mg/dl)***	9.8	(1.0)	9.3–10.6	9.6	(1.0)	9.2–10.6	(0.006)
Calcium urine (mg/dl)***	13.2	(2.2)	2.8–44.7	7.8	(1.9)	2.4–42.6	(0.001)
Calcium urine (mg/day)***	250	(1.5)	98–455	164	(2.0)	9–850	(0.0001)
Oxalate, urine (mg/day)***	47.6	(1.3)	29–74	41.2	(1.4)	13–83	(0.051)
Uric acid serum (mg/dl)***	6.3	(1.2)	4.2–8.9	6.5	(1.3)	3.5–9.8	—
Uric acid urine (mg/day)***	517	(1.8)	149–1434	504	(1.6)	125–1423	—
Urine volume (L/day)***	1.6	(1.4)	0.8–2.8	1.7	(1.6)	0.4–3.9	—
Calcium-oxalate index***	2.4	(1.7)	0.5–6.2	1.5	(1.8)	0.1–4.2	(0.0009)
Brushite index***	2.3	(2.4)	0.2–7.0	1.4	(2.7)	0.02–7.1	(0.048)

*Data on 208 persons completing core questionnaire.
**Data on 136 participants completing medical questionnaire.
***Geometric mean for 123 subjects providing blood and 24-hour urine.
⁺P-value stated only if p ≤ 0.20.

TABLE 4—Logistic Regression: Factors Predicting Kidney Stones*

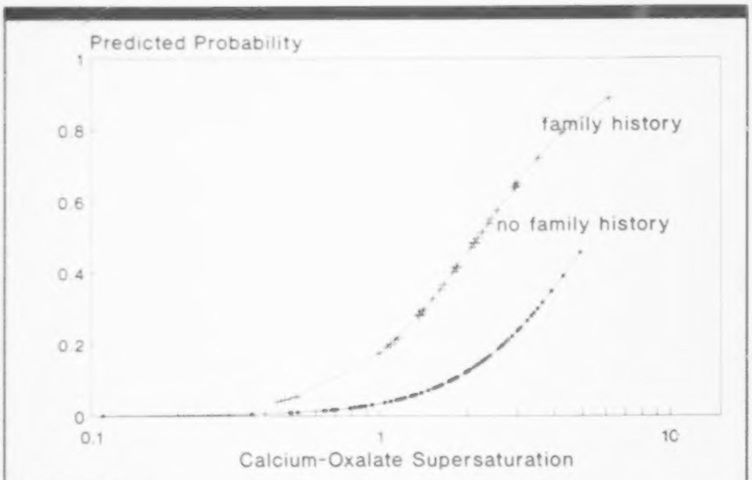
Risk Factors	Odds Ratio	SE	(95% CI)
Univariate Analyses			
Calcium-Oxalate Saturation Index			
Second tertile*	4.62	0.782	(1.0, 21.4)
Third tertile*	6.20	0.760	(1.4, 27.5)
Family history of stones	3.30	0.410	(1.5, 7.4)
Family history of infection	2.64	0.503	(1.0, 7.1)
Age 50–59 years	1.12	0.493	(0.4, 2.9)
Age ≥ 60 years	1.65	0.493	(0.6, 4.3)
Multivariate Analyses			
Calcium Oxalate Saturation Index			
Second tertile*	3.79	0.803	(0.8, 17.8)
Third tertile*	5.91	0.765	(1.3, 26.5)
Family History of Stones	3.51	0.425	(1.5, 8.1)
Age 50–59 years	1.62	0.505	(0.6, 4.4)
Age ≥ 60 years	2.21	0.505	(0.8, 5.9)

*Analysis based on 123 persons submitting blood and 24-hour urine sample.
**Mean scores for calcium-oxalate tertiles are 0.90, 1.79 and 3.01 for first, second, and third tertiles. Index is defined in text.

nary calcium was due to diet (milk, cheese, and meat) or to increased gastrointestinal absorption. However, we can exclude drinking water as a major source of calcium because municipal water supplies in the area have been monitored since 1982 and have unusually soft water due to low calcium.[22] Similarly, sunlight and its effect upon vitamin D cannot explain the measured values in our study, because the study was conducted in January. A second important determinant of CAOX was low urine volume. This cannot be attributed to incomplete urine collection, because it occurred in the presence of high 24-hour excretion of other stone risk factors. Low fluid intake is the most likely explanation for reduced urine volume and increased concentration. It is unclear why fluid intake should be low in these workers, although prohibition against alcoholic beverages in many areas of the southeast may play some role. Alcohol exerts a diuretic effect by suppressing the release of anti-diuretic hormone.[23] Consumption of moonshine liquor is negatively associated with stones in our study. The hypothesis that alcohol may protect against stones is plausible and deserves further evaluation.

Mildly elevated urinary oxalate (55–100 mg/24 hours)[24] occurred in 20 (16.5 percent) of the workers in our study. These levels are below those found in primary hyperoxaluria,[24] but may be consistent with dietary exposure. Foods commonly eaten in the southeast include several sources of oxalate (collards, mustard greens, kale, and ice tea).[25] Oxalate excretion contributed relatively little to urinary saturation with CAOX compared to hypercalcuria and low urine volume. However, the presence of oxalates in typical southern cuisine provides an intriguing hypothesis to be tested in future studies.

We cannot explain the low frequency of stones among the guards in our study. Guards were culturally different from the other workers. Twenty guards (46.5 percent) vs 10 (7.9 percent) of the other workers were retired military servicemen who had spent 20 or more years away from the area ($p < .0001$). Too few guards participated in the medical study to allow us to examine whether differences existed in their urine composition. The low participation occurred largely because guards



* defined in text

FIGURE 1—Probability of Stones by Family History and Calcium-Oxalate Supersaturation Index*

could not be released from duty to participate during working hours.

A limitation of our study is that the assessment of risk factors for stone-formation in urine is based upon a single 24-hour sample, obtained some years after the actual passing of stones. It is likely that excretion of most of these substances varies over time. Nevertheless, the strong association between current CAOX excretion and lifetime stone occurrence is consistent with the known importance of calcium and oxalate in stone formation. Excretion appears to be sufficiently stable to reflect a gradient of risk as current concentration increases. In summary, the study found an increased risk of urolithiasis and risk factors related to stone formation in male workers in eastern Tennessee. Future research should define the geographic range of increased kidney stone formation in the southeast and identify its causes. ☐

Acknowledgments

The authors thank the management, union, and workers of the three companies for their support. We thank Drs. Leslie Stayner and Victoria Wells for their help in the field testing, Brenda Lewis for laboratory assistance, Drs. Gary Diamond, Robert Goyer, Paul Morrow, and Richard Platt for peer review, Drs. Stacey FitzSimmons, Dana Flanders, and Charles Pak for their invaluable review of the manuscript, and Steven McGuire PhD and Judith Foulke, PhD, for serving as liaison project officers within the Nuclear Regulatory Commission (NRC). Support for the field and laboratory testing was provided by the NRC.

References

1. Boyce WH, Garvey FK, Strawcutter HE: Incidence of urinary calculi among patients in general hospitals, 1948–1952. JAMA 1956; 161:1437–1442.
2. Sierakowski R, Finlayson B, Landes R, Finlayson CD, Sierakowski N: The frequency of urolithiasis in hospital discharge diagnosis in the United States. Invest Urol 1978; 15:438–441.
3. Schey HM, Corbett WT, Resnick MI: Prevalence rate of renal stone disease in Forsyth County, North Carolina during 1977. J Urol 1979; 122:288–291.
4. Hiatt RA, Dales LG, Friedman GD, Hunkeler EM: Frequency of urolithiasis in a prepaid medical care program. Am J Epidemiol 1982; 115:255–265.
5. Johnson CM, Wilson DM, O'Fallon WM, Malek RS, Kurland LT: Renal stone epidemiology: A 25-year study in Rochester, Minnesota. Kidney Int 1979; 16:624–631.
6. Thun MJ, Schober S, Stayner L, Wells V: Health Hazard Evaluation Report, Nuclear Fuel Services, US Dept of Health and Human Services, Public Health Service, National Institute for Occupational Safety and Health, HETA-86-381-1934. Cincinnati: NIOSH, 1988.
7. Voegtlin C, Hodge HC (eds): Pharmacology and Toxicology of Uranium Compounds. Vols 1–4. New York: McGraw Hill, 1949.
8. Hodge HC, Stannard JN, Hursh JB (eds): Handbook of Experimental Pharmacology, Hefter-Heubner New Series, Vol XXXVI, Uranium, plutonium, and transplutonic elements. New York: Springer Verlag, 1973.
9. US Nuclear Regulatory Commission. Acute effects of inhalation exposure to uranium hexafluoride and patterns of deposition. NUREG 1CR-1045. Washington, DC: NRC, 1980.
10. Pak CYC, Skurla C, Harvey J: Graphic display of urinary risk factors for renal stone formation. J Urol 1985; 134:867–870.
11. Pak CYC, Hill K, Cintron NM, Huntoon C: Assessing applicants to the NASA flight program for their renal stone-forming potential. Aviation, Space, Environ Med 1989; 157–161.
12. Findlayson B: Calcium stones: Some physical and clinical aspects. *In:* David D (ed): Calcium Metabolism in Renal Failure and Nephrolithiasis. New York: J Wiley and Sons, 1979; 337.
13. US Department of Health and Human Services, Public Health Service, National Center for Health Statistics. Public use data tape documentation, Medical History, ages 12–74 years, Tape # 5020, NHANES 1976–80, pp 143–145. Hyattsville, MD: NCHS, 1985.
14. Rothman KJ, Boice JD: Epidemiologic analysis with a programmable calculator. Chestnut Hill, MA: Epidemiology Resources Inc, 1982; 13–14.
15. Kaplan EL, Meier P: Nonparametric estimation from incomplete observations. J Am Stat Assoc 1958; 53:457–481.
16. Coe FL, Kavalach AG: Hypercalciuria and hyperuricosuria in patients with calcium nephrolithiasis. N Engl J Med 1974; 291:1344–1350.
17. Sandler DP, Smith JC, Weinberg CR *et al*: Analgesic use and chronic renal disease. N Engl J Med 1989; 320:1238–1243.
18. Ljunghall S, Lithell H, Skarfors E: Prevalence of renal stones in 60-year-old men: A 10-year follow-up study of a health survey. Br J Urol 1987; 60:10–13.
19. Shuster J, Finlayson B, Schaeffer R, Sierakowski R, Zoltek J, Dzegede S: Water hardness and urinary stone disease. J Urol 1982; 128;422–425.
20. Parry ES: Sunlight and hypercalcuria. Lancet 1975; 1:1063–1065.
21. Shuster J, Finlayson B, Schaeffer RL, Sierakowski R, Zoltek J, Dzegede S: Primary liquid intake and urinary stone disease. J Chronic Dis 1985; 38;907–914.
22. Lauch RP, Sorg TJ: The occurrence and reduction of sodium in drinking water. J Am Water Works Assoc 1981; 50:256–265.
23. Andreoli TE: The posterior pituitary. *In:* Wyngaarden JB, Smith LH (eds): Cecil Textbook of Medicine. Philadelphia: WB Saunders Co, 1985; 1266–1273.
24. Williams H, Smith LH: Disorders of oxalate metabolism. Am J Med 1968; 45:715–732.
25. Fassett DW: Oxalates. *In:* Toxicants Occurring Naturally in Foods. Washington, DC: Committee on Food Protection, Food and Nutrition Board, National Research Council, National Academy of Sciences, 1973.

Benzodiazepine Anti-anxiety Agents: Prevalence and Correlates of Use in a Southern Community

Marvin Swartz, MD, Richard Landerman, PhD, Linda K. George, PhD, Mary Lou Melville, MD, Dan Blazer, MD, PhD, and Karen Smith, PhD

ABSTRACT

Background: Benzodiazepine anti-anxiety agents are the most widely prescibed psychotherapeutic drugs in the United States today. Recent evidence, however, suggests that their use may be decreasing.

Methods: We examine the population prevalence and correlates of use of benzodiazepine anxiolytics at the Duke site of the NIMH-sponsored Epidemiologic Catchment Area project.

Results: Bivariate analysis of use patterns for the drugs revealed demographic predictors similar to those reported in previous studies: increased likelihood of use by the elderly, Whites, women, the less educated, and the separated or divorced. Use is also associated with symptoms of psychic distress, negative life events, use of health care services, and diagnoses of affective disorder, agoraphobia with panic, and panic disorder. Age, sex, race, education, and marital status remain associated with non-hypnotic benzodiazepine use in a logistic regression analysis.

Conclusions: Multivariate analyses of these data indicate that when potential confounding factors are controlled, age, sex, race, education, and marital status are significantly related to benzodiazepine anxiolytic use but the effects of sex and education are mediated by intervening variables. Implications of these findings are discussed particularly in relation to high levels of use in the elderly. (*Am J Public Health* 1991;81:592-596)

Introduction

Benzodiazepine anti-anxiety agents are the most widely prescribed psychotherapeutic drugs in the United States today.[1] First introduced in 1960, these drugs rapidly achieved a lead position in the prescription drug market,[2] stimulating public and professional debate over appropriate psychotropic drug use.[3] Recent evidence, however, suggests that the prevalence and patterns of psychotropic use, especially those of benzodiazepine anxiolytics, may be changing and resulting in decreased use.[4,5] The first detailed population survey of psychotropic drug use, the National Household Sample in 1970–71,[6-9] found that 22 percent of American adults had used prescription psychotropic medication during the year 1969–70, with higher use among women and the elderly. The most commonly used drug group, "minor tranquilizer/daytime sedatives," was used by 8 percent of men and 20 percent of women. Use was greater in Whites and in divorced, separated, and widowed persons and the elderly.[6] In 1979, the National Survey of Psychotherapeutic Drug Use documented, using weighted data, that an estimated total of 8 percent of men and 13.7 percent of women had used benzodiazepine anxiolytics in the previous year with the greatest use among respondents ages 50-64.[2,10] Recent surveys of the St. Louis area population[11] and Great Britain[12] found lower overall rates of use but similar sex and age usage profiles.

Previous studies have relied primarily on bivariate analyses when describing patterns of drug use, failing to take into account the intercorrelations among predictors, including demographic, diagnostic, symptoms, and service use variables. In the present study we examine the prevalence and patterns of benzodiazepine antianxiolytic drug use in the Piedmont region of North Carolina during 1982–83, utilizing logistic regression analysis, which allows prediction of benzodiazepine use while introducing controls for potential confounding and mediating variables.

Method

The present paper reports results from Wave 1 of the Piedmont Health Survey, one site of the five-site National Institute of Mental Health Epidemiologic Catchment Area program (NIMH-ECA).[13] The sampling frame for the Piedmont Health Survey (PHS) was a five-county area in north central North Carolina, consisting of one urban county and four contiguous rural counties. The survey randomly sampled all housing units from segments throughout the catchment area, using the Kish method,[14] yielding 3,798 interviews usable for this study; further details of the study have been published elsewhere.[15] In this paper analyses are based on weighted data, taking into account household probability selection, nonresponse, and the 1980 census demographic profile of adults in the five-county catchment area. For significance tests, the data were downweighted to the original number of subjects, but weighting adjustments remain.

Diagnoses were obtained through use of the Diagnostic Interview Schedule

---

From the Psychiatric Epidemiology and Health Services Research Program, Department of Psychiatry, Duke University Medical Center. Address reprint requests to Marvin Swartz, MD, Box 3173, Duke University Medical Center, Durham, NC 27710. This paper, submitted to the Journal March 12, 1990, was revised and accepted for publication December 12, 1990.

(DIS), a highly structured interview designed for use by lay interviewers in epidemiological studies and capable of generating computer-based diagnoses for certain DSM-III disorders.[16] The diagnoses generated by the DIS were too numerous to include in a single regression equation. Preliminary analyses ascertained that an affective disorders summary measure (consisting of major depressive disorders, bipolar disorders, dysthymia, and bereavement), panic disorder, and agoraphobia with panic were most strongly related to benzodiazepine use. These measures were retained for subsequent analyses. The version of the DIS used in Wave 1 of this survey and other ECA sites did not include one anxiety disorders diagnosis, generalized anxiety disorder, employed at certain ECA sites in follow-up interviews.

In the present study psychotropic drug use refers to any use within the year prior to interview, regardless of amount or duration of use and was elicited by specific drug use probes aided by photographic drug identification cards. Other measures used for this analysis include a 29-item psychic distress scale analogous to the SCL-90,[17] and the number of negative life events during the year prior to the interview, measured by a scale of 20 major events of the type proposed by Holmes and Rahe.[18] Analyses of life events in the survey have been published elsewhere.[19,20] Interviewers also recorded the number of outpatient visits to physical and mental health providers during the six months prior to interview. The measure of outpatient health service utilization employed in this study is dichotomous, because the use vs non-use measure has more explanatory power than a continuous (number of visits) measure. Demographic variables used in this report include sex, age, race, education level, marital status, and urban vs rural residence. Income is excluded because preliminary analyses indicated that the relationship between anxiolytic use and income is weak.

The Appendix presents a six-stage causal model predicting the dichotomous measure of use vs nonuse of benzodiazepine anti-anxiety agents. A unidirectional causal process is assumed with variables in prior stages affecting those in subsequent stages. No causal direction is assumed for variables in the same stage. In the multivariate logistic regression analysis below, effects of the stage I variables are estimated first, and the equation is then expanded as groups of variables are entered by stage. This format yields an estimate of the effect of each variable net of all adjacent and prior variables and indicates whether the effects of prior variables are mediated by subsequent variables. The logistic regression procedure calculates maximum likelihood estimates for the parameters of a model that express the log odds of an event (here, drug use) as a simple linear model.[21,22]

Results

Table 1 presents the demographic profile for subjects participating in the survey.Table 2 focuses specifically on use of benzodiazepine anti-anxiety agents—predominantly diazepam and chlordiazepoxide—regardless of source. All other anxiolytics as well as all sedatives and hypnotics (benzodiazepine and other) are excluded. Table 2 presents a cross-tabular analysis of the weighted prevalence of benzodiazepine anxiolytic use among various demographic and other groups of interest. For two continuous variables, correlation coefficients are used: the correlation coefficient of drug use with number of negative life events is .09 ($p < .01$) and that for drug use with psychic distress symptoms is .20 ($p < .01$). Benzodiazepine anxiolytic use is more prevalent among women, older persons, Whites, and those with less education. The never married and married have significantly lower prevalence of use than the divorced and widowed. Respondents who have recently used either physical or mental health care services are much more likely to use these agents, as are those with a DIS diagnosis of affective disorder, panic disorder, or agoraphobia with panic.

Table 3 presents results of the logistic regression analyses. The "OR" coefficient in the table is the antilogged regression coefficient and estimates how changes in the independent variables multiply the odds of using benzodiazepine anxiolytics, holding other variables in the equation constant. Ninety-five percent confidence intervals are also given. The overall fit of the model is assessed by R statistic which is roughly analogous to the multiple correlation coefficient in ordinary least squares regression after correcting for the number of parameters estimated.[22]

In stage I, older persons and women are both more likely than others to use benzodiazepines, while Blacks are less likely. The gender effect decreases substantially in stages V and VI indicating that much of the initial sex difference is due to higher levels of psychic distress and health service utilization among women. The race effect changes very little across stages indicating no substantial effect of potential mediating variables. While the coefficients for age fluctuate across stages, they remain largely unmediated and substantial at stage VI. In stage II, increases across each of four levels of education (as indicated in Table 2) reduce the odds of benzodiazepine use by .9. This initial effect decreases in stages IV and V, indicating that those with more education use benzodiazepines less, in part, because they have fewer DIS diagnoses and psychic distress symptoms. Compared to the never married, the separated, divorced, and widowed are more likely to use benzodiazepine anxiolytics. These marital status effects are not mediated by varia-

TABLE 1—Demographic Profile of Community Subjects Participating in the Piedmont Health Survey (N = 3,798)

	No.[a]	%[b]	WTD[c]
Sex			
Male	1489	39	46
Female	2309	61	54
Race			
White	2399	63	62
Other[d]	1395	37	38
Age (years)			
18–34	1096	29	43
35–54	863	23	29
55–74	1414	37	23
75+	425	11	5
Marital Status			
Married with spouse	953	51	60
Separated/ divorced	480	13	11
Widowed	814	21	9
Never married	551	15	21
Educational Level (years)			
0–8	973	26	16
9–11	818	22	22
12–13	1062	28	34
14+	935	25	28
Residence			
Rural	1936	51	44
Urban	1962	49	56

[a] The total of some cells do not equal 3,798 because of missing data.

[b] The percents may not sum to 100 due to rounding.

[c] The weighted percentage adjusts the actual response percentage to take into account household probability selection, response rate, the elderly oversample and demographic characteristics of the 196,790 persons in the five-county area who were 18+ years of age. (Total five-county population, based on 1980 census data, was 269,863.)

[d] Other races are almost entirely Black.

TABLE 2—Bivariate Correlates of Benzodiazepine Anti-anxiety Agent Use, Piedmont Health Survey 1982–83

Variables	Total	Number Using Drug	WTD% Using Drug
Sex			
Males	1489	104	6.9
Females	2309	310	10.6[a]
Age (years)			
18–34	1096	50	3.8
35–54	863	99	11.5
55–74	1414	208	14.0
75+	425	57	13.6[a]
Race			
White	2399	317	11.5[a]
Non-White	1395	97	4.7
Marital Status			
Married	1953	197	9.3
Separated/divorced	480	70	11.4
Widowed	814	122	14.7
Never married	551	25	3.6[a]
Educational Level (years)			
0–8	973	124	11.3
9–11	818	111	11.8
12–13	1062	102	7.4
14+	935	77	7.2[a]
Residence			
Rural	1936	192	8.2
Urban	1862	222	9.4
DIS Diagnosis			
No Affective Dx	3638	364	8.2
1 Affective Dx	136	39	21.7
2+ Affective Dx	24	11	41.7[a]
No Agor. w/Panic Dx	3658	386	8.5
Agor. w/Panic Dx	40	10	33.1[a]
No Panic Disorder	3762	405	6.6
Panic Disorder Dx	22	6	39.2[a]
Health Service Utilization			
No Outpatient Physical Health Care (6 mos)	1619	84	3.9
Outpatient Physical Health Care (6 mos)	2150	325	13.2[a]
No Outpatient Mental Health Care (6 mos)	3450	301	7.0
Outpatient Mental Health Care (6 mos)	283	101	31.0[a]

[a] Significant at the .05 level or better. Where a particular set has more than two categories, significance test applies to the set.

bles entered in subsequent stages. Urban residence is not strongly associated with drug use. In stage III the number of negative life events is positively and substantially associated with drug use. Decreases in this effect in stages IV through VI indicate that the effect of negative life events is due primarily to the effects of life stress on DIS diagnoses, psychic distress symptoms, and increased health care utilization. In stages IV through VI, affective disorder, agoraphobia with panic, and panic disorder are all associated with increased benzodiazepine use, as are psychic distress and health care utilization. However as can be seen in the final model, effects of DIS diagnoses are mediated by psychic distress.

Discussion

Multivariate analysis of survey data indicates that when potential confounding factors are controlled, age, sex, race, education, and marital status are related to benzodiazepine anxiolytic use. Age, race, and marital status remain substantially unmediated by other factors while sex and education, previously thought to be predictors of anxiolytic use, exert their effects indirectly via their relationships with other predictors in subsequent model stages. The effect of education is mediated by intervening factors such as high levels of psychic distress or stressful life events. Women indeed use more anxiolytics, but probably because they either have or report more distress than men and/or are more likely to seek help. That is, net of the effect of increased levels of psychic distress and help-seeking among women, women are no more likely than men to receive and/or use anxiolytics, indicating less evidence for sex bias in prescribing practices and use than suggested in previous studies and the press.[11,23–25]

One of the most important findings in the present study relates to increased anxiolytic use in the elderly, many of whom are already medicated and/or cognitively impaired.[2] Benzodiazepine use in the elderly is a particular concern because the use of long half-life benzodiazepines such as the ones included in the present study is associated with an increased risk of hip fracture,[26] creating a potentially fatal complication of anxiolytic use in this population. High levels of use in the elderly raises concerns about the judicious use of these agents.

The differential rates of anxiolytic use by race, lower among Blacks even when predictors such as use of health services are controlled, cannot be explained further in the present analysis. These results do suggest possible racial bias in prescribing practices and/or different modes of help-seeking and alleviation of psychic distress among Whites vs Blacks. Prior studies have demonstrated that separated or divorced respondents have higher levels of life stress and psychic distress[9] and are more likely to use anxiolytics, presumably as a result of these stressors.[6,9] Our analyses suggest, however, that separated and divorced persons have a greater likelihood of drug use even net of negative life events and psychic distress, suggesting other explanatory or predispositional factors, not examined in the present analysis.

Finally these analyses raise questions about associations between symptoms, diagnosis, and appropriate psychotherapeutic drug use. There is evidence that the American public has become more conservative in its attitudes toward psychotropic drug use.[2,27] In fact, our preliminary data indicate that the majority of persons with high levels of anxiety in the community are not receiving psychotherapeutic medications; further analyses need to ex-

TABLE 3—Logistic Regression Effects Corresponding to Model in Appendix

	I OR (95% CI)	II OR (95% CI)	III OR (95% CI)	IV OR (95% CI)	V OR (95% CI)	VI OR (95% CI)
Intercept	0.04 —	0.05 —	0.03	0.03 —	0.02 —	0.01 —
Age 35–54	3.2 (3.8, 2.7)	2.7 (3.2, 2.2)	2.8 (3.4, 2.4)	3.0 (3.6, 2.5)	3.1 (3.8, 2.6)	3.1 (3.7, 2.5)
Age 55–74	4.0 (4.8, 3.4)	3.1 (3.7, 2.5)	3.4 (4.1, 2.8)	3.7 (4.5, 3.0)	4.0 (4.9, 3.3)	3.4 (4.2, 2.8)
Age 75+	3.6 (4.7, 2.8)	2.6 (3.5, 1.9)	2.9 (3.9, 2.1)	3.1 (4.3, 2.3)	3.4 (4.7, 2.5)	2.7 (3.7, 2.0)
Black	0.4 (0.4, 0.3)	0.4 (0.4, 0.3)	0.3 (0.4, 0.3)	0.3 (0.4, 0.3)	0.3 (0.4, 0.3)	0.3 (0.4, 0.3)
Female	1.5 (1.7, 1.3)	1.4 (1.6, 1.2)	1.4 (1.6, 1.3)	1.4 (1.6, 1.2)	1.3 (1.4, 1.1)	1.2 (1.4, 1.0)
Education		0.9 (0.9, 0.8)	0.9 (0.9, 0.8)	0.9 (1.0, 0.8)	1.0 (1.0, 0.9)	0.9 (1.0, 0.9)
Married		1.4 (1.8, 1.1)	1.5 (1.9, 1.1)	1.5 (1.8, 1.2)	1.5 (1.9, 1.2)	1.6 (2.0, 1.2)
Separated/divorced		2.2 (2.9, 1.6)	2.1 (2.8, 1.6)	2.1 (2.8, 1.6)	2.1 (2.8, 1.6)	2.3 (3.0, 1.7)
Widow		1.8 (2.4, 1.3)	1.7 (2.4, 1.3)	1.8 (2.4, 1.3)	1.7 (2.4, 1.3)	2.0 (2.7, 1.5)
Urban		1.3 (1.4, 1.1)	1.2 (1.4, 1.1)	1.2 (1.3, 1.0)	1.1 (1.2, 1.0)	1.1 (1.3, 1.0)
#Life stress			1.5 (1.6, 1.4)	1.4 (1.5, 1.3)	1.2 (1.3, 1.1)	1.1 (1.2, 1.0)
#Affect Dx				2.2 (2.6, 1.8)	1.2 (1.5, 1.0)	1.1 (1.3, 0.9)
Dx-agor/pan				2.8 (4.3, 1.8)	1.3 (2.0, 0.8)	1.3 (2.1, 0.8)
Dx-panic				4.6 (7.9, 2.6)	2.3 (4.0, 1.3)	1.3 (2.4, 0.8)
Psyc distress					1.2 (1.3, 1.2)	1.2 (1.2, 1.2)
Outpat phys						2.7 (3.2, 2.3)
Outpat ment						2.3 (2.7, 1.9)
Model:						
R	.26	.27	.30	.32	.36	.41

plore whether they are receiving other psychotherapeutic interventions. There is also a strong association between a DIS diagnosis of affective disorder and use of benzodiazepine anxiolytics. Concurrent use of antidepressants is not examined in the present study. However, Craig, *et al*, also found that surveyed women taking minor tranquilizers and sedatives included significantly more respondents with high depression scores, and that depressed respondents of both sexes tended to be taking anxiolytics or sedatives, and not antidepressants.[28] Beardsley, *et al*,[29] found that primary care practitioners provided 72 percent of all anxiolytic drug visits, but documented a psychiatric diagnosis in a minority of anxiolytic drug visits, raising questions about the specificity of anxiolytic use in primary care settings. Although anxious and depressive symptoms are often admixed, the present findings of high anxiolytic use among depressed respondents suggest that depressive symptoms are underrecognized. Major depressive episodes usually require specific antidepressant medication and psychotherapy, and treatment with benzodiazepine anxiolytics in the absence of more specific antidepressant therapy may postpone therapeutic response or even exacerbate symptoms.[23,30,31]

We have examined the current prevalence of benzodiazepine anxiolytic use in a Southern community, demonstrating that age, sex, race, education, and marital status are significantly associated with drug use, but that the effects of sex and education are mediated by intervening variables. In addition we have found benzodiazepine anxiolytic use to be significantly associated with several DIS diagnoses although the effects of diagnoses are largely mediated by psychic distress and use of health services. Future research on this population will focus on duration and frequency of anxiolytic use, use of other psychotropic drugs, and use of potentially interacting drug combinations. Finally, more detailed DIS diagnostic information gathered at follow-up of the survey cohort, including generalized anxiety disorder, will allow closer examination of the associations between benzodiazepine anxiolytic use and DIS diagnoses of affective and anxiety disorders. ☐

Acknowledgments

This study was funded by a grant to the Duke Department of Psychiatry from NIMH (MH 35386).

References

1. Baum C, Kennedy DL, Forbes MB, Jones JK: Drug use in the United States in 1981. JAMA 1984; 251:1293–1297.
2. Mellinger GD, Balter MB: Prevalence and patterns of use of psychotherapeutic drugs: Results from a 1979 national survey of American adults. *In:* Tognoni G, Bellantuono C, Lader M (eds): Impact of Psychotropic Drugs. Elsevier, North Holland Biomedical Press, 1981; 117–135.
3. Klerman GL: Psychotropic hedonism vs pharmacological calvinism. The Hastings Report 1972; 2:1–3.
4. Baum C, Kennedy DL, Forbes MB, Jones JK: Drug use and expenditures in 1982. JAMA 1985; 253:382–386.
5. Shader RI, Greenblatt DJ: Benzodiazepine overuse-misuse. J Clin Psychopharm 1984; 4:123–124.
6. Parry HJ, Balter M, Mellinger G, Cisin I, Manheimer D: National patterns of psychotherapeutic drug use. Arch Gen Psychiatry 1973; 28:769–783.
7. Uhlenhuth EH, Balter M, Lipman R; Minor tranquilizers. Arch Gen Psychiatry 1978; 35:650–655.
8. Mellinger GD, Balter MB, Parry HJ, Manheimer DI, Cisin IH: An overview of psychotherapeutic drug use in the United States. *In:* Josephson E, Carroll E (eds): Drug Use: Epidemiological and Sociological Approaches. New York: Hemisphere, 1974; 333–366.
9. Mellinger GD, Balter MB, Manheimer DI, Cisin IH, Parry HJ: Psychic distress, life crisis, and use of psychotherapeutic medications. Arch Gen Psychiatry 1978; 35:1045–1052.
10. Mellinger GD, Balter MB, Uhlenhuth EH: Prevalence and correlates of the long-term regular use of anxiolytics. JAMA 1984; 251:375–379.
11. Cottler LB, Robins LN: The prevalence and characteristics of psychoactive medication use in a general population study. Psychopharm Bull 1983; 19:746–751.
12. Dunbar GC, Perera MH, Jenner FA: Patterns of benzodiazepine use in Great Britain as measured by a general population survey. Br J Psychiatry 1989; 155:836–841.
13. Regier DA, Myers JK, Kramer M, Robins LN, Blazer DG, Hough RL, Eaton WW, Locke BZ: The NIMH Epidemiologic Catchment Area Program. Arch Gen Psychiatry 1984; 41:934–941.
14. Kish L: Survey Sampling. New York: John Wiley and Sons, 1965.
15. Blazer DG, George LK, Landerman R, Pennybacker M, Melville ML, Woodbury M, Manton KG, Jordan K, Locke B: Psychiatric disorders: A rural/urban comparison. Arch Gen Psychiatry 1985; 42:651–656.

16. Robins LN, Helzer JE, Croughan J, Ratcliff KF: National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics and validity. Arch Gen Psychiatry 1981; 38:381–389.
17. Derogatis LP, Lipman RS, Covi L: SCL-90: An outpatient psychiatric rating scale: Preliminary report. Psychopharm Bull 1973; 9:13–28.
18. Holmes TH, Rahe RH: The Social Readjustment Rating Scale. J Psychosom Res 1967; 11:213–218.
19. Hughes DC, Blazer DG, George LK: Age differences in life events: A multivariate controlled analysis. Int J Aging Human Devel 1988; 27:207–220.
20. Hughes DC, George LK, Blazer DG: Age differences in life event qualities: Multivariate controlled analyses. J Community Psychology 1988; 16:161–174.
21. Hanushek EA, Jackson JE: Statistical Methods for Social Scientists. New York: Academic Press, 1977.
22. Harrell FE: The LOGIST Procedure. *In:* Joyner S (ed): Supplemental Library User's Guide, 1983 Edition. Cary, NC: SAS Institute, Inc, 1983.
23. Rickels K: Use of antianxiety agents in anxious outpatients. Psychopharmacology 1978; 58:1–17.
24. Berg RH: The over-medicated woman. McCall's 1971; 67:109–111.
25. Rogers JM: Stimulus/response: Drug abuse by prescription. Psychol Today 1971; 5:16–24.
26. Ray WA, Griffin MR, Downey W: Benzodiazepines of long and short elimination half-life and the risk of hip fracture. JAMA 1989; 262:3303–3307.
27. Manheimer DI, Davidson ST, Balter MB, Mellinger GD, Cisin IH, Parry HJ: Popular attitudes and beliefs about tranquilizers. Am J Psychiatry 1973; 130:1246–1253.
28. Craig TJ, Van Netta PA: Current medication use and symptoms of depression in a general population. Am J Psychiatry 1978; 135:1036–1039.
29. Beardsley RS, Gardocki GJ, Larson DB, Hidalgo J: Prescribing of psychotropic medication by primary care physicians and psychiatrists. Arch Gen Psychiatry 1988; 45:1117–1119.
30. Hall RCW, Joffe JR: Aberrant response to diazepam: A new syndrome. Am J Psychiatry 1972; 129:738–742.
31. Greenblatt DJ, Shader RI: Benzodiazepines in Clinical Practice. New York: Raven Press, 1974.

APPENDIX
A Six-Stage Causal Model Predicting Benzodiazepine Use

I	II	III	IV	V	VI
Age	Education	Life Events	Affective Disorders	Psychic Distress	Utilize Outpatient Physical Health Services
Race	Marital Status		Agoraphobia with panic		
Sex	Urban Residence		Panic Disorder		Utilize Outpatient Mental Health Services
			Other Psychiatric Diagnoses		

The Influence of Three Mass Media Campaigns on Variables Related to Adolescent Cigarette Smoking: Results of a Field Experiment

Karl E. Bauman, PhD, John LaPrelle, PhD, Jane D. Brown, PhD, Gary G. Koch, PhD, and Connie A. Padgett, BA

ABSTRACT

Background: This paper reports findings from a field experiment that evaluated mass media campaigns designed to prevent cigarette smoking by adolescents.

Methods: The campaigns featured radio and television messages on expected consequences of smoking and a component to stimulate personal encouragement of peers not to smoke. Six Standard Metropolitan Statistical Areas in the Southeast United States received campaigns and four served as controls. Adolescents and mothers provided pretest and posttest data in their homes.

Results and Conclusions: The radio campaign had a modest influence on the expected consequences of smoking and friend approval of smoking, the more expensive campaigns involving television were not more effective than those with radio alone, the peer-involvement component was not effective, and any potential smoking effects could not be detected. (*Am J Public Health* 1991; 81:597-604)

Introduction

There is general agreement that cigarette smoking is the major preventable cause of death in the United States and that efforts to prevent smoking onset should be directed toward young people.[1]

Few studies have evaluated the influence of broadcast media on adolescent smoking.[2] Lewitt, Coate, and Grossman concluded that youth smoking rates were reduced by messages broadcast on radio and television.[3] The awareness of negative personal and social consequences of smoking increased among Minnesota adolescents after the introduction of campaigns that involved television, radio, and billboards.[4] Worden, *et al.*, found fewer reports of friend smoking, but no less smoking, among adolescents in a rural Vermont county that received a television campaign than in four matched control counties.[*] Flay, *et al.*, found no effects on smoking intention or behavior by a coordinated television and school-based prevention program.[5] Research on mass media campaign effects for behaviors other than smoking rarely involves adolescents (see, for example, Rice and Atkin[6]).

The purpose of this research was to evaluate the impact of mass media campaigns intended to prevent adolescent cigarette smoking.

Methods

The Mass Media Campaigns

The three mass media campaigns evaluated in this research are described in detail elsewhere.[7,8] They were developed with the guidance of behavioral science theory and research, one year of extensive formative research, and the principle that they could be implemented readily throughout the United States.

One campaign used eight 30-second radio messages that focused on seven expected consequences of smoking that are related to whether young people become regular smokers. We identify this campaign, and the areas in which it was implemented, as RADIO.

The second campaign (RPEER) was similar but also included a 60-second radio message that invited persons 12 to 15 years old to enter the "I Won't Smoke Sweepstakes." The purpose of the sweepstakes was to obtain names and addresses. We mailed entrants a brochure that asked them to talk to their friends about not smoking, to encourage their friends to pledge not to smoke, and to have their friends enter the sweepstakes. We offered a $20 incentive for recruiting five or more

---

[*] Worden JK, Flynn BS, McAuliffe TL, Sweeney RR, Secker-Walker RH: Using television to reach rural children with non-smoking messages. Paper presented at the American Rural Health Association Conference, Jeffersonville, VT, June 1982.

From the University of North Carolina, Chapel Hill, where Dr. Bauman and Ms. Padgett are with the School of Public Health, Department of Health Behavior and Health Education; Dr. LaPrelle is with the Department of Psychology; Dr. Brown is with the School of Journalism; and Dr. Koch is with the School of Public Health, Department of Biostatistics. Address reprint requests to Karl E. Bauman, PhD, Department of Health Behavior and Health Education, School of Public Health, the University of North Carolina, Chapel Hill, NC 27599-7400.

This paper, submitted to the Journal May 2, 1990, was revised and accepted for publication October 10, 1990.

entrants. The brochure also was mailed to friends they recruited.

The third campaign (RTVPEER) was similar to the second but included television broadcast of the sweepstakes offer and only three of the expected consequences messages.

The expected consequences messages were broadcast throughout November 1985, January 1986, and April 1986. The sweepstakes offer was broadcast during November 1985 and the brochures to encourage personal peer contact were mailed from January 1986 through February 1987. To simulate implementation of a national campaign, an advertising agency purchased broadcast time. The agency placed the messages at times expected to reach 75 percent of the intended audience four times during each of three four-week periods.

Design

The study design involved ten Standard Metropolitan Statistical Areas (SMSAs). Two SMSAs received each treatment condition and four served as CONTROL.

To minimize heterogeneity among study areas, only the 81 SMSAs in the Southeast United States were eligible for study. Homogeneity was increased further by excluding SMSAs with populations of fewer than 200,000 (n = 22) and more than 500,000 (n = 21) inhabitants, median age above 36 (n = 4), more than 90 percent White (n = 12), more than 22 percent with four or more years of college and ages 25 or older (n = 4), and density less than 100 or more than 300 per square mile (n = 2). We randomly eliminated SMSAs with overlapping broadcast areas until 10 SMSAs, located evenly throughout the Southeast United States, remained. We divided the Southeast into north, middle, and south, and randomly allocated the SMSAs to treatment conditions within each area. Cost and legal restrictions required two reassignments within area after random allocation. The final distribution of study SMSAs by treatment condition is CONTROL (Chattanooga, Tennessee; Columbia, South Carolina; Jackson, Mississippi; Savannah, Georgia), RADIO (Lakeland, Florida; Macon, Georgia), RPEER (Montgomery, Alabama; Roanoke, Virginia), and RTVPEER (Lexington, Kentucky; Mobile, Alabama).

Data Collection

Cluster sampling procedures identified probability samples of households within each SMSA in 1985.** Households were screened for adolescents ages 12 to 14. For the pretest surveys, conducted from April 1, 1985 through October 13, 1985, interviewers attempted to obtain data from all eligible adolescents and their mothers in the households. When more than one adolescent age 12 to 14 resided in a household, one was randomly selected to serve as a subject. Of the 2,534 adolescent subjects eligible for study in all SMSAs, 2,102 (83.0 percent) participated at pretest. The same subjects were asked to provide data again from April 16, 1987 to October 29, 1987, 11 to 17 months after the broadcasts ended and two to eight months after the mailings for the peer-involvement component ended. Of those who participated at pretest, 1,637 also participated at posttest and are included in the analyses reported here. Some pretest subjects could not be located at follow-up or had substantial missing information and are therefore lost to study. The numbers of subjects across SMSAs range from 132 to 232.

To assess overall attrition bias, we compared subjects who provided data only at pretest with those who also provided data at posttest on the intervening and smoking variables measured at pretest. The two groups did not differ on variables measuring smoking experimentation, subjective expected utility, smoking intention, friend approval of smoking, or friend encouragement of not smoking. Subjects lost to study, compared to subjects who provided both pretest and posttest data, were significantly more likely to be recent smokers (5.0 versus 3.1 percent), to be regular smokers (5.6 versus 3.1 percent), and to have high mean smoking intensity (1.22 versus 1.16).

While collecting posttest data from the panel subjects, we also gathered data from a cross-sectional sample of adolescents 14 to 16 years old and their mothers. Procedures were similar to those used to select subjects at baseline for the panel sample but we used clusters other than those in the panel and fewer subjects. We refer to this as the cross-sectional sample to distinguish it from the panel sample. The cross-sectional sample increased sample size and allowed assessment of testing effects.[9] Of the estimated 1,759 subjects eligible for the cross-sectional sample, 1,216 provided data.

Adolescent subjects averaged one hour to complete a self-administered questionnaire in their homes and provided alveolar breath and saliva samples to measure tobacco use. The mothers of most adolescents also completed a 25-minute self-administered questionnaire and provided alveolar breath samples.

Measures

The variables are listed in the Appendix. Data from the mother questionnaire measured parent education. All other measures are from data provided by the adolescents. Their derivation follows.

Adolescent subjects indicated the likelihood they would experience each of the seven expected consequences of smoking featured in the campaigns (bad breath, difficulty concentrating, loss of friends, trouble with adults, loss of appetite, increased fun, and increased relaxation) and how much they would like or dislike each consequence. We multiplied the responses for each consequence and summed the products to indicate the degree to which more positive or negative consequences of smoking were expected, with the more positive score favoring smoking. This measures subjective expected utility (SEU) for smoking (smoking SEU).[10] SEU for not smoking (nonsmoking SEU) was measured in the same way but the consequences were for not smoking rather than for smoking; more positive scores favored not smoking. A combined SEU (total SEU) was derived by subtracting nonsmoking SEU from smoking SEU; more positive scores on total SEU indicate greater preferences for smoking.

Decreased friend approval of smoking and increased friend encouragement of not smoking were hypothesized to be direct consequences of the peer-involvement component. To measure friend approval, subjects were asked how many of their three best friends would approve of their smoking. To measure friend encouragement, subjects were asked how often someone their own age had encouraged them to be a nonsmoker; response categories ranged from never to 10 or more times. To measure smoking intention, subjects were asked how sure they thought they would smoke in the next two years, with response categories ranging from certain to smoke to certain not to smoke.

Subjects provided alveolar breath samples to measure carbon monoxide (CO); a value ≥ 9.0 ppm indicated recent smoking.[11] Subjects were asked whether they had ever puffed on a cigarette. Subjects also were asked about their smoking intensity using a single questionnaire item that measured recency, frequency, and amount smoked.[12,13] The values ranged from never smoked a cigarette to currently smoke more than two packs a day. We also used the intensity measure to classify subjects as regular smokers if they usually smoked weekly or more often. To increase the validity of self-reports of smoking, subjects were told before beginning the questionnaires that the alveolar breath and saliva samples they gave would be used to measure their smoking.[14,15]

**Research Triangle Institute: Survey of Young Adolescent Smoking Behavior: Final report. 1986.

Analysis

Baseline data were used to create subsamples of subjects who were not regular smokers. Of the 1,637 panel subjects, the nine who did not report whether they had puffed on a cigarette were excluded from all analyses. The remaining subjects were divided into those who had not (N = 973) and those who had (N = 655) puffed on a cigarette. Of the adolescents who had not puffed on a cigarette, 22 with missing information on CO or with CO ≥ 9 ppm were excluded; the remaining 951 subjects are nonexperimenters. Of the adolescents who had puffed on a cigarette, 127 who were regular smokers, had CO ≥ 9 ppm, or had missing information were excluded; the remaining 528 are experimenters. The total sample includes experimenters (N = 528), nonexperimenters (N = 951), regular smokers (N = 48), and subjects with insufficient information for classification (N = 110).

Each SMSA was assigned average scores based on the information provided by subjects within the SMSA. Multiple regression analysis for repeated measures was the principal statistical method for inferences concerning treatment differences for changes over time by SMSA, i.e. treatment by time interaction.[16] We did not include control variables in analyses using the SMSA as the unit because these units had small sample size (n = 10), were randomized, and were relatively homogeneous with respect to previously stated population characteristics. Analyses with the individual as the unit used logistic regression for dichotomous dependent variables and linear regression for continuous dependent variables. These latter analyses examined effects for treatment, SMSA nested in treatment, and control variables from the pretest. We included the control variables related to an intervening or dependent variable and to pretest differences between treatments, attrition bias, or missing data substitution. For an intervening or dependent variable at posttest, we controlled for the corresponding pretest measure of the variable.

Results

Independent Variables

Message reach and frequency were estimated by the advertising agency using standard estimating procedures with Arbitron and Nielsen data for radio and television, respectively.[17] The expected consequences messages reached 81 percent of the intended audience an average of 4.5 times during each of the three four-week periods that they were broadcast. The sweepstakes offer in RPEER reached 79 percent of the audience an average of four times. In RTVPEER, messages reached 90 to 94 percent of the intended audience by a combination of radio and television, and they were seen or heard an average of 14 times during the entire period of broadcast. Young people ages 12 to 15 years surveyed by telephone the month after the final campaign messages were broadcast reported higher levels of reach.[18]

Based on sweepstakes entry and US Census data, 21 to 24 percent of the adolescents 12 to 15 years old in RTVPEER entered the sweepstakes; 97.8 percent of them responded to the encouragement of others rather than to the broadcast messages.[8] Nine to 15 percent of the adolescents in RPEER entered the sweepstakes, and 94.8 percent of them responded to their friends rather than to the broadcast.[8]

Intervening Variables

Between pretest and posttest, all intervening variables changed significantly in the expected direction of becoming more favorable toward smoking (Table 1). The p values in Table 1 for the time by treatment interactions indicate whether the change in the intervening variables between 1985 and 1987 varies significantly by treatment. There were significant time by treatment interactions for smoking SEU (nonexperimenters and total sample), nonsmoking SEU (experimenters only), and total SEU (nonexperimenters and total sample).

In Table 2, we show the 1985 and 1987 values of smoking SEU for the total sample, by treatment condition, and measures of treatment effect for change between 1985 and 1987. Smoking SEU increased less in RADIO and RTVPEER than in CONTROL, increased by the same amount in RADIO and RTVPEER, and increased by the same amount in RPEER and CONTROL. These findings suggest that RADIO and RTVPEER had the intended effects of decreasing smoking SEU. These effects of generally 4.0 correspond to about one-third of the baseline standard deviation of smoking SEU, which is comparable to a shift between the median and the 60th percentile. Thus, the campaign effect size is modest rather than large. The findings were similar for the other SEU measures and samples for the statistically significant treatment by time interactions shown in Table 1.

The findings in Table 2 suggest that the peer-involvement component in RPEER might have had undesirable effects, but this seems unlikely given the lower participation in the peer-involvement component in response to RPEER than to RTVPEER. However, to address the possibility of undesirable effects attributable to the peer-involvement component, we disaggregated subjects within RPEER and RTVPEER according to their participation in the peer-involvement campaign. Participants had entered the sweepstakes or reported on their posttest questionnaires that they had been personally contacted to enter the sweepstakes; all other subjects are nonparticipants. We conducted analyses similar to those shown in Tables 1 and 2 using the intervening variables as dependent variables because the campaigns were designed to influence them directly. Within both RPEER and RTVPEER areas, participants did not differ from nonparticipants in their 1985 to 1987 change in SEU or friend approval of smoking, but participants were more likely than nonparticipants to report an increase in encouragement by friends not to smoke. These findings suggest that the peer-involvement component did not produce undesirable effects. The absence of other treatment conditions, such as one with both radio and television broadcast of expected consequence messages and no peer involvement component, precludes analyses that might clarify these findings.

Changes in the SMSAs between 1985 and 1987 other than the campaigns might have produced the higher increase in smoking SEU in RPEER than in RTVPEER and RADIO shown in Table 2. For example, if adolescent use of smokeless tobacco increased more in RPEER than in RADIO and RTVPEER and adolescent use of smokeless tobacco is related to smoking SEU, then the differential change in smokeless tobacco use might explain the smoking SEU patterns. Using the individual as the unit of analysis, we identified intervening

TABLE 1—Multiple Regression Analysis for Repeated Measures of Time, Treatment, and Time by Treatment Effects for Intervening Variables

		Means		p		
Variables	Subsample	1985	1987	Time	Treatment	Time by Treatment
Smoking SEU	Nonexperimenters	−20.41	−16.41	.001	.540	.014
	Experimenters	−15.62	−12.61	.037	.896	.391
	Total sample	−17.88	−14.72	.004	.748	.061
Nonsmoking SEU	Nonexperimenters	21.79	17.79	.001	.928	.386
	Experimenters	18.27	12.91	.001	.107	.029
	Total sample	19.96	15.65	.001	.760	.321
Total SEU	Nonexperimenters	−42.21	−34.46	.001	.772	.028
	Experimenters	−33.89	−25.47	.002	.373	.120
	Total sample	−37.84	−30.33	.001	.632	.092
Friend approval of smoking cigarettes	Nonexperimenters	.33	.65	.001	.700	.003
	Experimenters	.90	1.14	.010	.836	.257
	Total sample	.60	.89	.001	.860	.078
Friend encouragement of not smoking cigarettes	Nonexperimenters	1.06	1.28	.023	.196	.742
	Experimenters	1.21	1.48	.002	.341	.288
	Total sample	1.14	1.39	.004	.240	.778
Intention not to smoke cigarettes	Nonexperimenters	4.62	4.42	.001	.975	.304
	Experimenters	4.23	3.91	.005	.865	.449
	Total sample	4.40	4.16	.001	.896	.248

Notes: SEU = subjective expected utility
n = 10
Source: Panel sample

TABLE 2—Descriptive Statistics, Measures of Effect, and Treatment Comparisons for Smoking Subjective Expected Utility for Total Sample

						Treatment and Control Change	
Treatment	n	Statistics	1985 Mean	1987 Mean	1985 vs 1987 Change	Difference	(95% CI)
CONTROL	4	Estimate	−18.74	−14.16	4.58	Reference	
		SE	.70	.62	.93		
RADIO	2	Estimate	−16.64	−16.28	.36	−4.22	(−8.16, −.28)
		SE	.99	.87	1.31	1.61	
RPEER	2	Estimate	−19.22	−13.80	5.42	0.84	(−3.10, 4.78)
		SE	.99	.87	1.31	1.61	
RTVPEER	2	Estimate	−16.08	−15.20	.88	−3.70	(−7.64, .24)
		SE	.99	.87	1.31	1.61	
Comparison among Groups	p-value		.14	.24	.06	.06	

SE = Standard Error

variables other than SEU, and the control variables as measured at pretest, that were related to nonsmoking SEU, smoking SEU, or total SEU at posttest. They were dependent variables in analyses conducted to identify statistically significant time by treatment interactions that parallel the findings involving smoking SEU and total SEU. There were only two statistically significant and parallel interactions, but control for the involved variables did not account for the patterns in Table 2. The greater increase in SEU in RPEER than in RADIO and RTVPEER remains unexplained.

There were significant time by treatment interactions for friend approval of smoking for nonexperimenters and for the total sample (Table 1). The 1985 and 1987 values of friend approval for the total sample, by treatment condition, and measures of treatment effect for change between 1985 and 1987 are in Table 3. These findings suggest that RADIO caused less increase in friend approval of smoking relative to the control condition. The estimated effect of RADIO corresponds to a modest reduction of 7 percent in the number who reported that one or more of their friends approve of smoking. The findings were the same for nonexperimenters.

There were no significant time by treatment effects that involved smoking intention or friend encouragement of smoking.

Cigarette Smoking

Table 4 shows the findings with cigarette smoking as dependent variables. The means of all smoking variables changed significantly between 1985 and 1987 in the expected direction of more smoking. However, none of the p values for the time by treatment interactions were statistically significant. The data are consistent with the conclusion that the campaigns did not influence smoking.

There were substantial within-treatment differences in smoking between SMSAs that must be recognized because they precluded the ability to identify campaign effects for smoking if there were any to be

TABLE 3—Descriptive Statistics, Measures of Effect, and Treatment Comparisons for Friend Approval of Smoking Cigarettes for Total Sample

						Treatment and Control Change	
Treatment	n	Statistics	1985 Mean	1987 Mean	1985 vs 1987 Change	Difference	(95% CI)
CONTROL	4	Estimate	.56	.87	.31	Reference	
		SE	.05	.06	.05		
RADIO	2	Estimate	.72	.81	.09	−.22	(−.43, −.01)
		SE	.07	.09	.07	.09	
RPEER	2	Estimate	.58	.99	.41	.10	(−.11, .31)
		SE	.07	.09	.07	.09	
RTVPEER	2	Estimate	.60	.90	.30	−.01	(−.22, .20)
		SE	.07	.09	.07	.09	
Comparison among Groups	p-value		.39	.56	.08	.08	

SE = Standard Error

TABLE 4—Multiple Regression Analysis for Repeated Measures of Time, Treatment, and Time by Treatment Effects for Dependent Variables

		Means		p		
Variables	Subsample	1985	1987	Time	Treatment	Time by Treatment
Smoking experimentation	Nonexperimenters	—	.37	—	—	.906
	Total sample	.40	.57	.002	.410	.637
Regular smoking	Nonexperimenters	—	.05	—	—	.782
	Experimenters	—	.18	—	—	.907
	Total sample	.03	.14	.001	.916	.148
Recent smoking	Nonexperimenters	—	.03	—	—	.563
	Total sample	.03	.08	.001	.762	.157
Smoking intensity	Nonexperimenters	—	1.23	—	—	.910
	Total sample	1.16	1.50	.001	.876	.778

Note: n = 10
Source: Panel sample

detected. We began the study with the assumption that 4 percent of adolescent subjects in each study SMSA would be regular smokers and that SMSA changes in smoking within treatment condition would be similar. About 4 percent of our subjects were regular smokers in 1985. However, the range across SMSAs was .6 percent to 5.2 percent. The variation across SMSAs in the other smoking measures, and changes in smoking, also varied substantially across SMSAs. Most importantly, smoking varied substantially among SMSAs within treatment condition. Table 5 shows the treatment-control differences in smoking and the intervening variables that would have been required to achieve statistical significance at the .10 level for powers of .50 and .80 given the SMSA variation in smoking within treatment conditions in our data; the actual differences are also shown. The relatively large error variance for SMSAs within treatment groups clearly precluded detecting campaign effects for smoking. Therefore, whether the campaigns influenced smoking cannot be determined with acceptable certainty from our data.

Related Findings

We also used the 1987 cross-sectional data to examine campaign effects. For each SMSA, we assigned pretest values of the intervening and dependent variables in the panel data to the cross-sectional data. We then conducted multiple regression analysis for repeated measures with a sample size of 20 for the analysis: 10 panel and 10 cross-sectional SMSAs. The main statistic of interest was time by treatment interaction. The conclusions about campaign effects were generally the same as those when only the panel data were used.

Having subjects with and without baseline measures allowed us to identify effects due to the pretest. To assess this, as part of the analyses described in the preceding paragraph, we evaluated the time by treatment by data source (panel versus cross-sectional) interactions involving each intervening and dependent variable. The findings suggest that there were no testing effects.

We also conducted analyses to determine whether campaign effects for smoking varied across selected subgroups by testing for time by treatment by subgroup interactions with multiple regression analysis for repeated measures. The 10 variables used to create the subgroups, each divided at the median except when categorical, were: gender, age, smoking by best friend, smoking by mother, school curricula on smoking, total SEU, alcohol use, parent education, number of hours spent listening to the radio, and number of hours spent watching television. This analysis revealed no pattern that could be interpreted as campaign effects for smoking, and the conclusions for intervening variables remained the same.

TABLE 5—Required and Observed Differences between Treatment and Control

Variables	Required[a] Power		Observed		
	.50	.80	RADIO	RPEER	RTVPEER
Nonexperimenters					
Smoking experimentation	.19	.25	.01	.06	.00
Regular smoking	.06	.07	.02	.02	.00
Recent smoking	.03	.04	.01	.02	.02
Smoking intensity	.21	.28	.01	.03	.06
Total SEU[b]	4.38	5.79	8.96	.13	5.07
Smoking SEU[b]	2.63	3.48	6.34	.60	4.22
Nonsmoking SEU	2.99	3.96	2.63	.37	.86
Smoking intention	.19	.25	.04	.21	.01
Friend approval of smoking[b]	.08	.10	.27	.04	.00
Friend encouragement of not smoking	.38	.50	.12	.21	.01
Total Sample					
Total SEU[b]	5.25	6.94	5.86	2.38	5.29
Smoking SEU[b]	3.05	4.04	4.04	.98	3.60
Nonsmoking SEU	3.04	4.02	1.73	1.44	1.66
Smoking intention	.19	.25	.02	.20	.02
Friend approval of smoking[b]	.16	.22	.21	.09	.01
Friend encouragement of not smoking	.29	.38	.01	.15	.05

Note: SEU = subjective expected utility

a) Differences required to identify effects 50 and 80 percent of the time given study inter-SMSA variance.

b) Overall ANOVAs significant at alpha = .10 (n = 10).

Finally, we conducted analyses of the relationship between treatment and the intervening and dependent variables when using individuals rather than SMSAs as the unit of analysis. This procedure could artificially increase statistical detection of differences by ignoring intra-SMSA correlation among subjects.[19] In these analyses, we controlled for the pretest value of each control variable that was related to the posttest value of the intervening or dependent variable, the pretest value of the intervening or dependent variable being examined, and variables indicating attrition bias and effects from missing-data substitution. The findings were generally the same as when the SMSA was the unit of analysis. SEU effects were limited to RADIO and RTVPEER and no effects for cigarette smoking were detected.

Discussion

SEU and friend approval of smoking appear to have been influenced by the media campaigns. RADIO was as effective for these variables as any of the other more expensive campaigns. The peer-involvement component was not effective. We detected no effect for reducing the onset of smoking, and any potential effect for smoking remains to be demonstrated.

The substantial variation in smoking within treatment conditions is a major methodological limitation of this research because it precluded determining whether the campaigns influenced cigarette smoking. A consideration supporting the possibility of some campaign influence on smoking is that the campaigns appear to have influenced SEU and friend approval of smoking, variables that are related to the onset of smoking.[7,8] In addition, the campaigns featured characteristics that media scholars consider necessary for campaign effects, such as extensive formative research, content based on behavioral science theory, high quality message production, actors with characteristics admired by the audience, and repeated exposure to the campaigns by most of the intended population.[20-22] Earlier studies have found that mass media campaigns can influence behavior.[6]

Some contend that substantial impact by mass media campaigns requires their implementation with other types of interventions, such as community organization efforts or school-based curricula.[2,23] Our study was designed to determine whether the media by themselves are influential. This is the most common and practical application of mass media in public health and, unlike multiple-component approaches, is capable of ready distribution on a national level. However, this also precludes our knowing whether adding other types of prevention programs to the campaigns we designed and implemented would have produced larger effects.

To increase homogeneity of SMSAs within and across treatment conditions, we limited our study to SMSAs in the Southeast US, included only SMSAs similar on social and demographic characteristics, stratified the Southeast by area, and randomly allocated SMSAs to treatment conditions within area. Still, substantial variation in adolescent smoking precluded our determining whether there were campaign effects for smoking. We might have avoided the problem by studying a much larger sample of SMSAs and perhaps matching them for baseline smoking behavior before random allocation to treatment conditions.

Information on other interventions that might have been implemented to influence adolescent smoking in our study areas would have been useful for attempting to explain and control the SMSA variation in smoking. We assume that school-based programs would have been the only potentially significant organized activities implemented to reduce adolescent smoking during the time of our study; we controlled for school curricula and many other variables at baseline when using the individual as the unit of analysis. The substantial variation in adolescent smoking by SMSA, and our findings on effect size for intervening variables, suggest that more powerful interventions than the mass media campaigns we implemented might be necessary to demonstrate community-level behavioral effects for programs designed to prevent adolescent smoking.

We designed the peer-involvement component to influence perception of friend approval of smoking. However, the findings suggest that only RADIO, which did not have a peer involvement component, influenced friend approval. Perhaps RADIO influenced peer approval because two of the expected consequences messages focused on peers. Or, perhaps the radio messages were projected to their friends because they are like the young people who recorded them. The reason this effect did not also occur in response to RPEER and RTVPEER was not determined.

Given the centrality of personal influence to many behavioral science and mass communication theories, it is significant that the two campaigns with the peer-involvement component, RPEER and RTVPEER, were not more effective than RADIO. Perhaps the personal contact produced by the peer-involvement component was too superficial for impact, perhaps this approach is less powerful than commonly believed, or perhaps effects would have appeared for younger or older people. We do not have the data required to assess these possibilities.

Implementing the campaigns in 1985 for a broadcast reach of 75 percent of the United States population 12 to 17 years old under the conditions of our field experiment, excluding development and research expenses, would have cost $1,843,000 for RADIO, $12,866,000 for RPEER, and $28,944,000 for RTVPEER. Although radio is the less expensive medium, television is the broadcast medium that receives the most attention by those who design, implement, and evaluate mass media campaigns. Perhaps the glamour associated with television explains why there have been few recent studies of health-oriented radio campaigns. Radio use increases relative to television use during adolescence,[24] and our findings suggest that radio should be given more attention in campaigns to influence adolescent smoking and by research on campaign effectiveness.

Although findings that demonstrate program impact for intervening variables such as SEU and friend approval but not for behavior can sometimes be dismissed readily, one can also argue that they merit attention. SEU for smoking and friend approval for smoking have been considered by many to be precursors of smoking.[7,8] Our findings suggest that RADIO did influence SEU and friend approval. These findings, together with the capability of radio campaigns to be implemented readily on a national scale or to other large populations at lower cost than virtually any other type of intervention, support its potential role for health promotion among adolescents. Whether adolescent smoking would be influenced by our radio campaign or by others, however, has yet to be shown. The failure of the more expensive components, television broadcast and peer involvement, to influence the intervening variables suggests that these components might be unworthy of further consideration, at least as they were implemented in our research.

Our findings cannot be generalized to all applications of the mass media to smoking prevention. We might have found other effects if we had done things differently, such as focused on younger or older adolescents, implemented the campaigns in smaller or larger communities, or also implemented smoking prevention curricula in schools. The effects also might have been different if we had used other theories for guidance and if the style and format of our messages had been different. Flay, *et al*,[25] and Worden *et al*,[26] describe other approaches to using the mass media to prevent adolescent smoking. Finally, our findings cannot be generalized to the many public service campaigns that rely solely on donated broadcast time and fail to reach most of their intended audiences. ☐

Acknowledgments

This research was supported by Grant CA38392 from the National Cancer Institute of the National Institutes of Health. Elizabeth S. Bryan and Lynn A. Fisher contributed substantially to the methods used in this research and to the design of the mass media campaigns; John M. Sweeney played a major role in the creation of the mass media messages; Elizabeth Turney conducted some of the analyses reported in this paper. This paper reflects comments on earlier drafts by Susan Ennett, Robert L. Flewelling, Dr. Vangie A. Foshee, and Harm Hospers. Dr. [illegible] Boyd, Dr. Thomas J. Glynn, and researc[illegible] funded by the same program of the National Cancer Institute provided advice and assistance throughout the conduct of this research.

References

1. US Department of Health and Human Services: Reducing the health consequences of smoking: 25 years of progress. A report of the Surgeon General. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. DHHS Pub. No. (CDC) 89-8411, Prepublication version. Rockville, MD: Author, January 11, 1989.
2. Flay BR: Selling the smokeless society: Fifty-six evaluated mass media programs and campaigns worldwide. Washington, DC: American Public Health Association, 1987.
3. Lewitt EM, Coate D, Grossman M: The effects of government regulation on teenage smoking. J Law Econ 1981; 24:545–569.
4. Minnesota Department of Health: The Minnesota Tobacco-Use Prevention Initiative, January 1987–December 1988: A report to the 1989 Legislature. Minneapolis: Minnesota Department of Health, 1989.
5. Flay BR, Hansen WB, Johnson CA, *et al*: Implementation effectiveness trial of a social influences smoking prevention program using schools and television. Health Educ Res 1987; 2:385–400.
6. Rice RE, Atkin CK (eds): Public Communication Campaigns, 2nd Ed. Newbury Park, CA: Sage, 1989.
7. Bauman KE, Brown JD, Bryan ES, Fisher LA, Padgett CA, Sweeney JM: Three mass media campaigns to prevent adolescent cigarette smoking. Prev Med 1988; 17:510–530.
8. Bauman KE, Padgett CA, Koch GG: A media-based campaign to encourage personal communication among adolescents about not smoking cigarettes: Participation, selection and consequences. Health Educ Res 1989; 4:35–44.
9. Solomon RL: An extension of control group design. Psychol Bull 1949; 46:137–150.
10. Bauman KE, Fisher LA, Bryan ES, Chenoweth RL: Antecedents, subjective expected utility, and behavior: A panel study of adolescent cigarette smoking. Addict Behav 1984; 9:121–136.
11. Bauman KE, Koch GG: Validity of self-reports and descriptive and analytical conclusions: The case of cigarette smoking by adolescents and their mothers. Am J Epidemiol 1983; 118:90–98.
12. National Cancer Institute: Smoking, tobacco, and cancer program: 1985 report. NIH Pub. No. 86-2687. Bethesda, MD: National Institutes of Health, Public Health Service, US Department of Health and Human Services, 1986.
13. Pechacek TF, Murray DM, Luepker RV, Mittelmark MB, Johnson CA, Shutz JM: Measurement of adolescent smoking behavior: Rationale and methods. J Behav Med 1984; 7:123–140.
14. Bauman KE, Dent CW: Influence of an objective measure on self-reports of behavior. J Appl Psychol 1982; 67:623–628.
15. Murray DM, O'Connell CM, Schmid LA, Perry CL: The validity of smoking self-reports by adolescents: A reexamination of the bogus pipeline procedure. Addict Behav 1987; 12:7–15.
16. Morrison DF: Multivariate Statistical Methods, 2nd Ed. New York: McGraw Hill Book Company, 1986; 128–166.
17. Alcalay R, Taplin S: Community health campaigns: From theory to action. *In:* Rice RE, Atkin CK (eds): Public Communication Campaigns, 2nd Ed. Newbury Park, CA: Sage, 1989; 124–127.
18. Brown JD, Bauman KE, Padgett CA: A

validity problem in measuring exposure to mass media campaigns. Health Educ Q 1990; 17:301-306.

19. Murray DM, Hannan PJ: Planning the appropriate analysis in school-based drug-use prevention studies. J Consult Clin Psychol 1990; 58:458-468.
20. Backer TE: Health professionals' and mass media campaigns to prevent AIDS and drug abuse. Counsel Hum Devel 1988; 20:2-10.
21. DeJong W, Winsten JA: The use of mass media in substance abuse prevention. Health Aff 1990; 9:30-46.
22. Rogers EM, Storey JD: Communication campaigns. *In:* Berger CR, Chaffee SH (eds): Handbook of Communication Science. Newbury Park, CA: Sage, 1987; 817-846.
23. Flora JA, Maccoby N, Farquhar JW: Communication campaigns to prevent cardiovascular disease: The Stanford community studies. *In:* Rice RE, Atkin CK (eds): Public Communication Campaigns, 2nd Ed. Newbury Park, CA: Sage, 1989; 233-252.
24. Brown JD, Childers KW, Bauman KE, Koch GG: The influence of new media and family structure on young adolescents' television and radio use. Comm Res 1990; 17:65-82.
25. Flay BR, Brannon BR, Johnson CA, *et al*: The television school and family smoking prevention and cessation project: 1. Theoretical basis and program development. Prev Med 1988; 17:585-607.
26. Worden JK, Flynn BS, Geller BM, *et al*: Development of a smoking prevention mass media program using diagnostic and formative research. Prev Med 1988; 17:531-558.

APPENDIX—Intervening, Dependent and Control Variables

Intervening

Smoking subjective expected utility
Nonsmoking subjective expected utility
Total subjective expected utility
Friend approval of smoking
Friend encouragement of not smoking
Smoking intention

Dependent

Smoking experimentation
Smoking intensity
Regular smoking
Recent smoking

Control: Background

Gender
Race
Age
Parent education
Years residence in area
Father presence

Control: Parent and Friend Smoking

Mother approval of adolescent smoking
Smoking by mother
Smoking by three best friends
Smoking by best friend

Control: General Social and Psychological

Popularity
Rejection
Egotism
Social passivity
Reputation
Femininity
Self-deprecation
Depression
Boredom
Sensation-seeking
Deviance
Rebelliousness
Religious involvement
Risk-seeking
Locus of control
Impulsivity
Closeness to mother
Peer versus parent influence

Control: Media Use

Number of hours listen to radio
Number of hours watch television

Control: Exposure to Antismoking Interventions

Advertisements
School curricula

Control: Other

Smokeless tobacco use
Alcohol use*

---

*Variable used only in analyses to determine if campaign effects varied across subgroups.

A Comparison of Injury Death Rates in China and the United States, 1986

Guohua Li, MB, MPH, and Susan P. Baker, MPH

ABSTRACT

Background: The importance of injury as a public health problem is not well recognized in many developing countries. Data have recently become available on injury mortality in China.

Method: We compared Chinese injury data based on a 100 million population segment for 1986 with data for the United States.

Results: The age-adjusted death rate from all injuries for China exceeds the rate for the US (69.0 vs 61.3 per 100,000). The US has higher death rates from motor vehicle crashes, fires, and homicide; China has greater mortality from drowning, poisoning, falls, and suicide. Especially noteworthy in China are the high drowning rates among young children and the elderly and the high suicide rates in rural areas among young adults and the elderly.

Conclusion: Injury is an important public health problem in China, exceeding in many respects the problem in the United States. It is urgent for China to place high priority on injury research and prevention. (*Am J Public Health* 1991;81:605–609)

Introduction

Injury is now well recognized in the United States and other developed countries as an important public health problem, the leading cause of death from the first year of life to age 44.[1] Most efforts and achievements are limited to the US and other industrial nations. In developing countries, injury research is still in its infancy.

The People's Republic of China, with about one-fifth of the world's population, reflects many aspects of other developing countries. The authors compared the injury death rates in China and the United States to determine whether the great differences between China and the US in their social, economic, and cultural background are associated with major disparities in injury death rates.

Methods

China does not have a complete nationwide disease and injury surveillance system. Data on mortality have become available since the early 1980s with the development and extension of the disease surveillance points (DSP) in its 30 provinces, covering about 100 million population or about 10 percent of the total population of the country. The 1986 DSP injury data for China were provided by the World Bank and are published in summary form.[2] Data for the US come from the National Center for Health Statistics (NCHS).[3] Both sets of data are based on the Ninth Revision of the International Classification of Diseases.

Results

Crude and Age-adjusted Death Rates

The US has a higher crude death rate than China for all injuries combined, but the Chinese rate is higher when adjusted to the 1986 US population (Table 1). The death rates from falls and suicide for China increase significantly after adjustment, as rates from these causes are very high for the elderly and the American population is older than China's. Chinese age-adjusted death rates from falls, drowning, and suicide are two to four times the US rates.

For all injuries, the US demonstrates much greater gender differences. In US males, the age-adjusted rate is 2.7 times the rate for females. In China, the male rate is only 1.1 times the female rate.

Percent of Injuries by Cause

The top three causes of injury death in China are suicide, motor vehicle crashes, and drowning, which account for 33 percent, 16 percent, and 14 percent of all injury deaths, respectively (Figure 1). Motor vehicle crashes, suicide, and homicide are the leading causes of injury death in the US, accounting for 31 percent, 20 percent, and 14 percent of all injury deaths, respectively. The prominence of falls, poisoning, and suffocation is similar in the two countries.

Death Rates by Cause and Area

In both China and the US, urban areas have higher death rates from poisoning and falls while rural areas have higher death rates from fires/burns and drowning. For these causes, the urban/rural pattern differs markedly between the coun-

---

From the Injury Prevention Center, Johns Hopkins School of Hygiene and Public Health. Address reprint requests to Guohua Li, MB, MPH, Injury Prevention Center, Johns Hopkins School of Hygiene and Public Health, 624 N. Broadway, Baltimore, MD 21205. This paper, submitted to the Journal May 31, 1990, was revised and accepted for publication October 10, 1990.

tries. In the US, homicide rates are very high in large cities and motor vehicle death rates are high in rural areas. Suicide rates, on the other hand, are similar in urban and rural areas of the US, while in China the rates in rural areas are almost three times the urban rates (Table 2).

TABLE 1—Adjusted Death Rates* from Injuries by Cause and Sex in China and US, 1986

Cause	Male		Female		Total		
					China		
	China	US	China	US	Crude	Adjusted	US
Motor Vehicle	12.7	28.4	5.9	11.0	8.5	9.5	19.4
Poisoning	3.7	3.5	2.8	1.3	2.6	3.3	2.4
Falls	9.2	5.1	14.7	4.4	5.8	12.0	4.7
Fires and Burns	1.6	2.5	1.7	1.5	1.0	1.7	2.0
Drowning	10.1	3.3	6.0	0.7	7.7	8.1	2.0
Suffocation	1.9	1.7	1.7	1.4	1.7	1.8	1.5
Suicide	17.7	20.6	24.3	5.4	17.6	21.1	12.8
Homicide	1.9	14.1	0.9	4.2	1.4	1.4	9.0
All Injuries	71.4	90.6	65.6	33.6	54.1	69.0	61.3

*Adjusted to the 1986 US population.

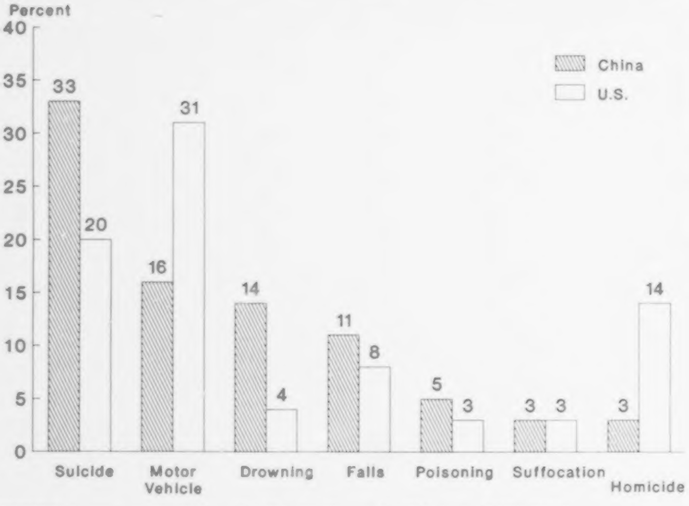


FIGURE 1—Percent of Injury Deaths by Cause in China and US, 1986

TABLE 2—Crude Death Rates from Injury by Cause and Area, in China and US*

Cause	China (1986)		US (1980–1986)	
	Urban	Rural	Central Cities	Remote Rural
Motor Vehicle	8.4	8.6	17.0	29.2
Poisoning	2.7	2.4	3.0	1.9
Falls	6.7	4.6	6.6	3.9
Fires/Burns	0.7	1.4	2.6	3.6
Drowning	4.0	12.7	2.0	5.9
Suffocation	0.6	3.1	0.4	0.6
Suicide	10.0	27.7	13.6	12.7
Homicide	1.6	1.2	17.7	5.4
All Injuries	41.4	71.0	73.4	80.7

*Source: Baker SP, et al.[10]

Death Rates by Age and Sex

All Injuries: China has higher death rates at ages 0–14 and above 55 years, while the US has higher death rates at ages 15–54 (Table 3). The greatest disparity is at the two extremes of age; for children under one year the death rate in China is almost five times the US rate, and for age 85 and older, the rate is 2.5 times the US rate. For both countries there is a peak at ages 20–24 and a sharp rise after age 65. For males, the peak at ages 20–24 is more extreme in the US, where death rates exceed the Chinese rates from ages 15 to 59. In contrast, death rates for Chinese females are consistently higher than those for US females, except at ages 25–29. There is a pronounced peak in the death rate at ages 20–24 for Chinese females, but not for US females.

Motor Vehicle Crashes: Motor vehicle death rates in the US are more than double the rates in China for both males and females ages 10 to 29 (Figure 2). A peak at ages 15–24 characterizes the American death rates for both sexes. Otherwise, death rates increase fairly steadily throughout the life span in both countries.

Poisoning: High-risk populations for poisoning in China are infants (under one year) and the elderly (above 60 years), and the peak appears at age 85 and over for both males and females. In the US, the death rate also increases in the elderly, but is highest in males in their thirties.

Falls: In both China and the US, the high-risk population for falls is the elderly. Falls are the leading cause of fatal injury among ages 65 and above for both males and females in the two countries. The death rates for males at age 85 and above are 301.7 and 163.1 per 100,000 in China and the US, respectively. For females, the corresponding figures are 404.4 and 121.8. The Chinese rates are more than four times the US rates for children less than 15 years old.

Fires and Burns: Americans have higher death rates from fires and burns before age 70 than Chinese. However, Chinese rates are higher after age 70 than American rates. Notably, the US death rate from fires and burns under age five is more than twice the Chinese rate for both males and females.

Drowning: Drowning is the third leading cause of injury death in China. The drowning rates at ages 1–4 for China are 45.4 and 34.4 deaths per 100,000 for males and females, respectively (Figure 3), seven to 10 times the American rates. Except for males ages 25–29, China has higher death rates from drowning for both males and females than the US.

Suffocation: Suffocation accounts for about 3 percent of all injury deaths. In infants, the rates in China are 19 times and 31 times the US death rates for males and females, respectively. Above age 85, on the other hand, the US rates are 42 times and 12 times the Chinese rates for males and females, respectively. In most other age groups the two countries share very low death rates from suffocation.

Suicide: Suicide is the leading cause of injury death in China and ranks second in the US. For males, China has lower death rates before age 60 and higher rates after age 60 than the US (Figure 4). For Chinese females, the suicide rate is more than 10 times the US rate at ages 15–24 and after 60, and more than twice the US rate for ages 25–59.

Homicide: In contrast to suicide, homicide is a major killer only for Americans. For each sex and age group, the US has higher homicide mortality (Figure 5). In both countries, male death rates are two to three times the rates for females. For the US, homicide rates are highest in the twenties and then decline until the sixties. For children less than 5 years old, the homicide rate in the US is more than four times the rate in China.

Discussion

Injury mortality data for China have not previously been described in detail. The general profile of injury death rates in China differs profoundly from the US. China's age-adjusted mortality from all injuries surpasses that of the US, even though its crude rate is lower than the US rate. In particular, China has higher death rates from falls, drowning, and suicide, and the US has higher rates from motor vehicles, fires and burns, and homicide.

Although the population-based death rate from motor vehicles in the US is twice as high as in China, there is evidence that China does not have safer transportation than the US. On average, China has about 15 motor vehicles per 1,000 population, which is less than a fortieth of the US ratio.[4,5] Its death rate per 100,000 motor vehicles is more than 10 times the rate of the US. The mortality from motor vehicles in China has been rising in the last 10 years[4] while declining in the US and other industrial nations.[6,7] With industrialization of China, death rates will continue to rise and the tragedy experienced by developed countries will be repeated, unless China pays enough attention to highway safety research and learns lessons from developed nations.

TABLE 3—Death Rates from All Injuries by Age and Sex, in China and US, 1986

		<1	1–	5–	10–	15–	20–	25–	30–	35–	45–	55–	65–	75–	85+
Male	China	141	63	36	24	42	68	44	54	58	56	79	143	285	671
	US	33	27	16	23	102	141	121	107	93	87	88	110	209	422
Female	China	141	48	15	12	39	57	29	32	33	37	56	108	277	692
	US	30	19	9	10	34	34	30	27	26	28	32	46	95	221

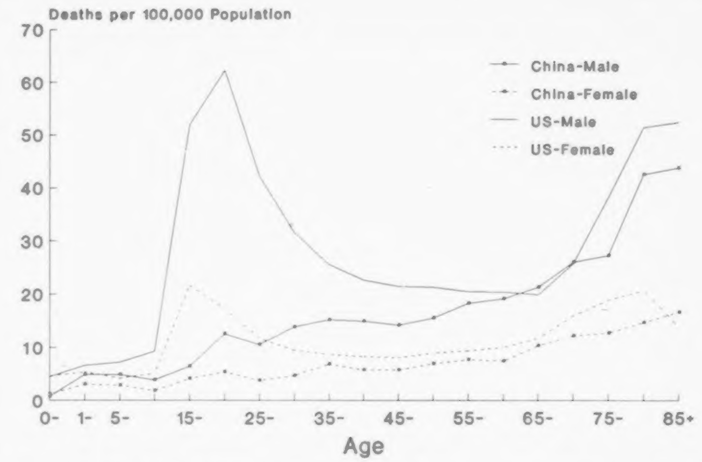


FIGURE 2—Death Rates from Motor Vehicles by Age and Sex in China and US, 1986

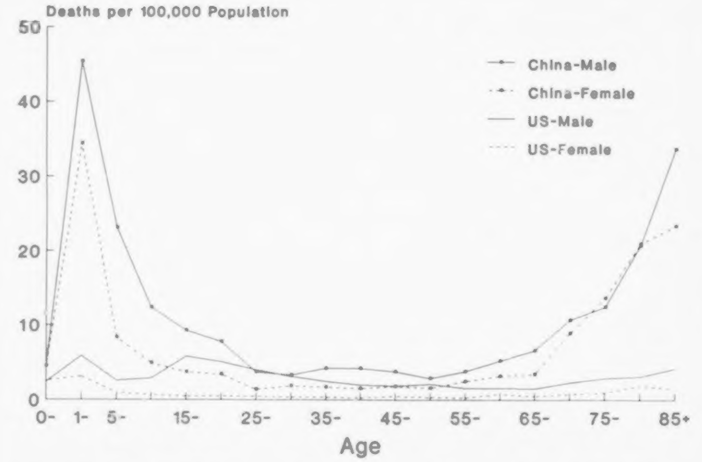


FIGURE 3—Death Rates from Drowning by Age and Sex in China and US, 1986

The very high suicide rate in China observed among ages 15–24, and among females age 60 or older, reflects many so-

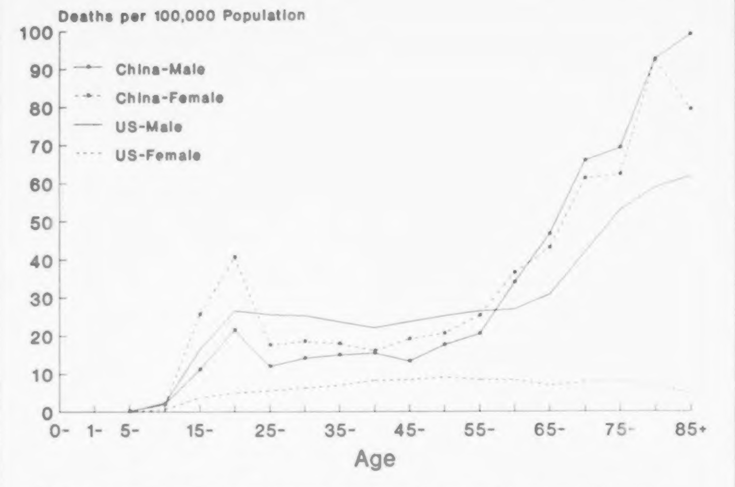


FIGURE 4—Death Rates from Suicide by Age and Sex in China and US, 1986

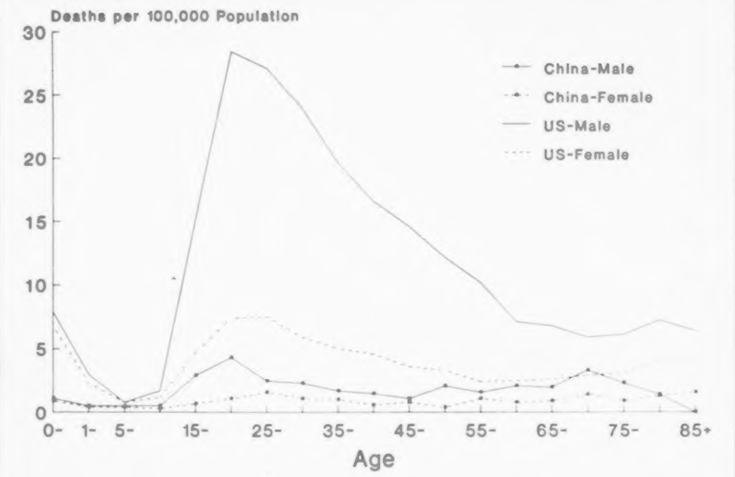


FIGURE 5—Death Rates from Homicide by Age and Sex in China and US, 1986

cial and cultural problems that China is facing. The motivation and cause of suicide may be greatly different between Chinese and Americans. For young Chinese females, especially those living in rural areas, marriage problems and poverty may be the major causes of the high suicide rate. For Chinese females at ages 20–24, the suicide rate is as high as the motor vehicle death rate for the American males in the same age group. In this age group, the suicide rate among females in rural areas is five times the rate in urban areas (78.3 vs 15.9/100,000). Similarly, for Chinese males ages 20–24, rural areas have four times the suicide rate as urban areas (40.7 vs 9.9/100,000). For Chinese elderly, poverty, long-term disease, and the decrease of traditional family relationships may contribute to the extremely high suicide rates. In the US, on the other hand, the suicide rate is highest in large cities and lowest in very rural areas. Because of the very complicated factors influencing suicide, China, as other countries, may have even greater difficulty in reducing its high suicide rates than in controlling its rising mortality from motor vehicles.

In contrast to suicide, the US has much higher homicide rates than China for each sex. Firearms are the weapon most commonly used in American homicide.[8,9] The law prohibiting individual ownership of firearms and virtual eradication of drug abuse may be major contributors to the relatively low homicide rate in China. On the other hand, even the non-firearm homicide rate in the US is substantially higher than China's total homicide rate, suggesting the important contribution of other factors.

Drowning is the third leading cause of injury death in China. Its major victims are young children and the elderly. Drowning rates in the US are highest at ages 1–3,[10] but are much lower than in China. One of the most effective interventions is barriers for swimming pools, which could reduce about 70 percent of childhood drownings in pools.[11] As drowning occurs in ponds, lakes, rivers, and other natural settings in China, to prevent and reduce the incidence is very difficult. In some rural areas of south China, drowning is the leading killer of children at ages 1–7.[12]

Among all injuries, falls exhibit the most similar profile in the two countries, although China has higher death rates at each age. Studies show that effective measures can reduce the incidence for children.[13,14] The higher death rates in China from falls in the elderly may reflect poor access to medical care, since mortality from falls often results from complications following relatively minor injury and has been greatly reduced in the US in recent years.[10]

Although the injury data for China are reasonably valid, some problems affect our comparison. First, about 80 percent of the Chinese population live in rural areas, but the rural population in the disease surveillance points sample accounts for only 43 percent. This should greatly underestimate the total death rate from all injuries in China, since the rate is much higher in rural areas than in urban areas. In particular, it leads to an underestimation of China's suicide and drowning rates, which are especially high in rural areas. Second, as ICD-9 has not been widely used in China, some misclassifications probably exist, and it is difficult to determine the degree of misclassification. Even so, our results suggest that injury is a serious public health problem in China that merits high priority. Special studies and injury surveillance systems that will provide details on the circumstances of injury are needed, followed as soon as possible by implementation of programs for effective injury prevention. ☐

Acknowledgments

We thank J. Richard Bumgarner of the World Bank for providing the data on injuries for China that stimulated development of this paper. This research was supported by Centers for Disease Control Grant No. R49/CCR302486-05 from the Division of Injury Epidemiology and Control, Center for Environmental Health and Injury Control.

References

1. National Research Council: Injury in America: A Continuing Public Health Problem. Washington, DC: National Academy Press, 1985.
2. World Health Organization: World Health Statistics Annual. Geneva: WHO, 1989.
3. National Center for Health Statistics: Vital and Mortality Statistics. Hyattsville, MD: US Public Health Service, 1986.
4. Li GH: Epidemiology of Fatal Traffic Accidents in Wuhan City, Hubei Province, 1979–85. Beijing (Conference Documents): Beijing International Academic Conference on Epidemiology, 1989.
5. Waller JA: Injury Control: A Guide to the Causes and Prevention of Trauma. Lexington, MA: D.C. Health, 1985.
6. World Health Organization: World Health Statistics Annual. Geneva: WHO, 1980.
7. World Health Organization: World Health Statistics Annual. Geneva: WHO, 1988.
8. Baker SP, Teret SP, Dietz PE: Firearms and the public health. J Public Health Policy 1980; 1:224–229.
9. Federal Bureau of Investigation: Crime in the United States: Uniform Crime Reports. Washington, DC: US Department of Justice, 1987.
10. Baker SP, O'Neill B, Ginsburg M, Li G: The Injury Fact Book, 2nd Ed. New York: Oxford University Press, in press.
11. Baxter L: Infant Drowning in Swimming Pools/Spas. Washington, DC: Household Structural Products Program, US Consumer Product Safety Commission, 1984.
12. Liang HC: Social Medicine. Hunan: Hunan Press of Science and Technology, 1988.
13. Spiegel CN, Lindaman FC: Children can't fly: A program to prevent childhood morbidity and mortality from window falls. Am J Public Health 1977; 67:1143–1147.
14. Bergner L, Mayer S, Harris D: Falls from heights: A childhood epidemic in an urban area. Am J Public Health 1971; 61:90–96.

Core Curriculum on Tuberculosis Available through CDC

A training document on tuberculosis is now available through the Centers for Disease Control (CDC). The document was developed as part of the National Tuberculosis Training Initiative (NTTI) which was implemented in 1989 by the Division of Tuberculosis Control of CDC, the American Thoracic Society, and over 22 national professional medical and nursing associations including the American Public Health Association. Increasing the knowledge of health care providers about tuberculosis is a critical element in the Strategic Plan for the Elimination of Tuberculosis in the United States, which was recently endorsed by APHA.

The "Core Curriculum on Tuberculosis" contains information on epidemiology, transmission and pathogenesis, screening and prevention, diagnosis, treatment, infection control, and public health aspects of tuberculosis. It includes information on managing patients with coexisting human immuno-deficiency virus (HIV) infection. It is intended for use in developing educational programs as well as serving as a reference manual for health care providers caring for persons with or at risk of clinically active tuberculosis and tuberculous infection.

Copies may be obtained by writing to: Ms. Landis Brown, Technical Information Services, Center for Prevention Services, Centers for Disease Control, Mailstop E-06, Atlanta, GA 30333. Members of the American Public Health Association should identify themselves as such.

A slide series has been developed to accompany the "Core Curriculum on Tuberculosis." This series may be obtained on loan (and duplicated) from your State tuberculosis control program.

Comparison of Two Growth Charts in Lesotho: Health Workers' Ability to Understand and Use Them for Action

ABSTRACT

Background: Two growth monitoring charts widely used for growth monitoring in Africa (the Road-to-Health (RTH) and the Growth Surveillance (GS)) were compared in order to assist the Government of Lesotho to decide on an appropriate national growth chart.

Methods: Thirty-four health workers were taught and tested on the RTH during a first week of training and on the GS during a second week (the RTH-GS group), while the order was reversed for another 25 trainees (the GS-RTH group). The health workers were trained and tested on their ability not only to use and interpret the two charts, but also to make the right decisions about specific actions to be taken when growth faltering occurs.

Results: There was no difference between scores to the RTH and GS charts after one week of training. After the second week of training, the scores to the RTH chart improved and became better than those to the GS chart. The scores to the GS test did not increase with previous knowledge of the RTH chart.

Conclusions: For this reason and others discussed in the paper, the RTH chart was recommended for nationwide use in Lesotho. The adoption of this recommendation was facilitated by the close involvement in this research of public and private agencies responsible for growth monitoring in Lesotho. (*Am J Public Health* 1991;81:610–616)

Marie T. Ruel, PhD, David L. Pelletier, PhD, Jean-Pierre Habicht, PhD, John B. Mason, PhD, C. S. Chobokoane, PhD, and Arapang P. Maruping, MD, MPH

Introduction

Growth monitoring has been promoted by international agencies and local governments as one of the principal activities to combat malnutrition among young children in developing countries.[1,2] Growth charts are used

- as an educational and promotional tool, i.e., to make the dynamic process of growth visible to both health workers and mothers and to motivate them to take action to improve or maintain the child's nutritional status[3] and
- as a screening tool to allow the early detection of growth faltering in children and the targeting of appropriate interventions.[4]

Four conditions are necessary for growth charts to be useful for either educational or screening purposes. First, the child's age and weight data must be reasonably accurate. Second, health workers must be able to understand and interpret growth charts properly. Third, once growth faltering is detected, health workers must be able to identify the appropriate actions that need to be taken. Fourth, mothers must also be able to understand the growth charts and the messages provided by the health workers. The second and third issues are addressed in the present paper and the fourth one is discussed elsewhere.[5]

In Lesotho, a small kingdom surrounded by the Republic of South Africa, the concern about health workers' and mothers' ability to understand growth charts originated from the widespread use of two different charts throughout the country. The Ministry of Health, which administered approximately one-third of the Primary Health Care clinics, was using the World Health Organization (WHO)[6] version of the Road-to-Health chart (Figure 1). The remaining clinics were administered by the Catholic Relief Services and used the Growth Surveillance system.[7] This system consists of two charts: the Master Chart (Figure 2A), retained at the clinic for statistics compilations and the Growth Surveillance chart (Figure 2B), taken home by mothers.

In Lesotho, both charts had strong proponents. Those advocating the Growth Surveillance chart emphasized the fact that this chart had been developed in Africa and was more socially and culturally acceptable for African health workers and mothers. Although the superiority of this chart over the Road-to-Health had never been tested, it was assumed that, for Africans, a straight flat line would be a better graphical representation of adequate growth (regular weight increase) than an ascending slope. On the other hand, the proponents of the Road-to-Health chart felt that African mothers were not different from other mothers in understanding a positive slope as representing adequate growth and a negative slope as a sign of weight loss. Furthermore, proponents of the Road-to-Health chart felt that it was easier for health workers to learn and

---

Address reprint requests to Marie T. Ruel, Institute of Nutrition of Central America and Panama (INCAP), Carretera Roosevelt, Zone 11, Apartado 1188, Guatemala, Guatemala, Central America. Drs. Pelletier and Habicht are with the Division of Nutritional Sciences, Cornell University, Ithaca, NY; Dr. Mason is Technical Secretary, ACC/SCN, World Health Organization, Geneva; Dr. Chobokoane is with the Food and Nutrition Coordinating Office, Prime Minister's Office, Maseru, Lesoto; Dr. Maruping is WHO Medical Officer, Harare, Zimbabwe. This paper, submitted to the Journal March 13, 1990, was revised and accepted for publication December 17, 1990.

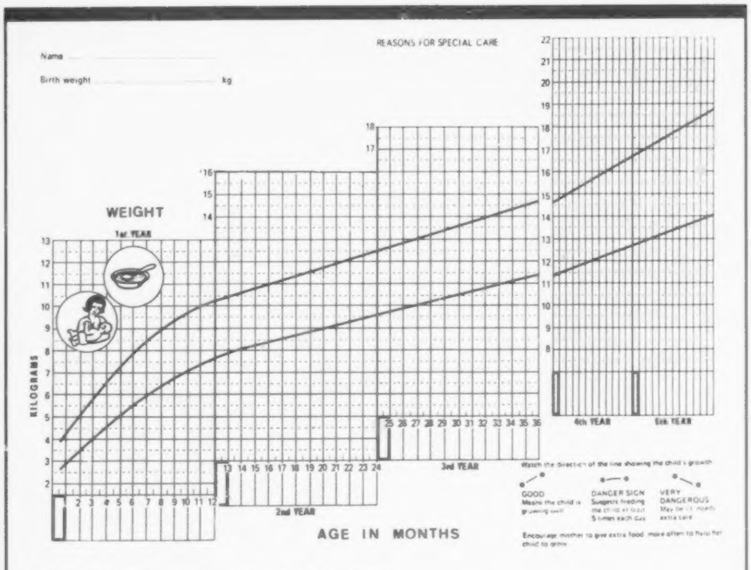


FIGURE 1—The Road-to-Health (RTH) Chart

interpret than the Growth Surveillance chart. UNICEF fostered this study to resolve these differences of opinion.

A Steering Committee, composed of members from various institutions involved in growth monitoring in Lesotho (see Appendix I for the list of participating institutions), was formed to advise the Government of Lesotho in the choice of a single growth chart to be used uniformly throughout the country. The final decision was to be based on the result of two studies, one that assessed health workers' ability to understand and use the charts for decision making (present study) and one that compared mothers' understanding of the two charts.[5] This Committee was involved in discussions and decisions about the various aspects of the research, including setting the objectives, choosing the experimental design, and developing the questionnaires and testing instruments. It also participated in the development of "Guidelines for Action", i.e. guidelines for decisions to be taken from the results of a weighing (Appendix II). Finally, the Steering Committee played a crucial role in facilitating implementation of the study in the field and in reviewing the results and discussing the inferences and implications for the country.

The objectives of the present study were to evaluate health workers' understanding and skills in using and interpreting the two growth charts after one week of training in each one, and in using the information for counseling and referral decisions according to the actions the Committee felt were indicated.

Methods

Study Sample and Design

Fifty-nine health workers from four hospital training centers of Lesotho were included in the study. Each participant was taught the two charts on two consecutive weeks.

A cross-over design was used to examine and take into account possible carry-over effects occurring when the learning of a chart taught second is made easier by the knowledge of the first chart. Therefore, two different treatment sequences were used and training centers were randomly assigned to either one: Hospitals A and B were taught the Growth Surveillance chart first and the Road-to-Health chart second (GS-RTH group), while Hospitals C and D were reversed and taught the Road-to-Health chart first and the Growth Surveillance chart second (RTH-GS group).

Growth Charts Used

The two charts compared were the RTH (Figure 1) and the GS (Figure 2B) chart. On the RTH chart, weight in kilograms forms the vertical axis and age of the child in months (ranging from 0–60 months) is on the horizontal axis. Two curves are printed on the chart and delimit the "Road-to-Health" zone. The upper one represents the median values for the reference population (50th percentile of the National Center for Health Statistics standards for boys[6]) and the lower one represents the NCHS third percentile for girls.[6] The GS chart contains the *percentage* of median weight-for-age (using the Harvard standards)[7] on the vertical axis and age in months on the horizontal one. Thus a straight line on this chart represents a child whose growth continues at a constant percentage of the standard weight-for-age. A green horizontal band (black in Figure 2B) delimits the so-called zone of "adequate growth" which corresponds approximately to the "Road-to-Health" on the RTH chart. A Master Chart (Figure 2A) must be used before plotting the data on the GS chart to find the corresponding percentage of standard weight-for-age for each child.

The decisions to be taken after weighing a child as developed by the Steering Committee (Appendix II) were also taught and used in the final test to assess the health workers' ability to use the charts for decision-making.

Teaching and Testing Procedures

One experienced teacher, who had been trained by the Catholic Relief Service and the Ministry of Health staff on the GS and the RTH charts, respectively, conducted all the teaching sessions. The format of the lecture on each chart had been standardized so that the amount of time spent on each aspect of the chart was the same for both. All lectures were taped and reviewed by the project director (MR) to ensure uniformity of content and lack of bias in the teaching.

On their first day, all participants were tested on their knowledge and understanding of both charts (baseline test) and information was collected on their previous exposure to the charts and work experience in primary care clinics. Each week of training in each chart consisted of approximately six hours of theory and six hours of practice. The training was done in a classroom setting using case histories. The trainees were instructed on how to calculate age given the date of birth (using a standard technique), how to plot hypothetical weights on growth charts, how to interpret growth curves, and what decisions to make (see Appendix II: Guidelines for Action).

The participants' knowledge, interpretation of the chart, and ability to make the appropriate decisions as prescribed by the Guidelines for Action were tested at the end of each training week. The final tests (one for the RTH and one for the GS)

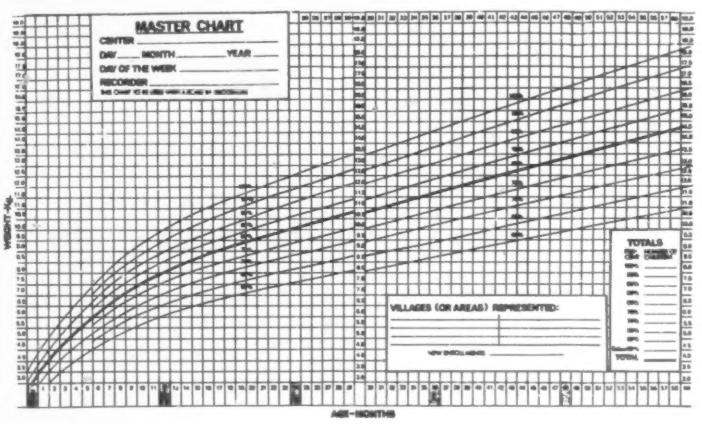


FIGURE 2A—The Master Chart

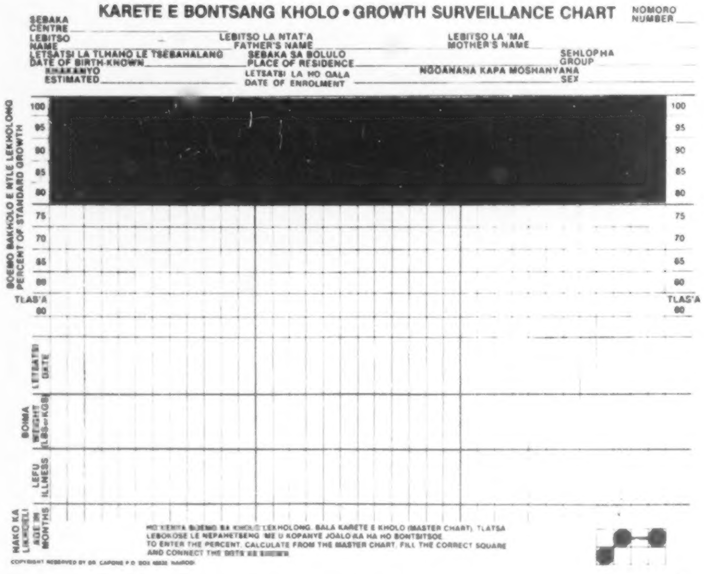


FIGURE 2B—The Growth Surveillance (GS) Chart

were very similar in their format and degree of difficulty. They were composed of case histories with questions on: age calculation, plotting of weights on the charts, interpretation of growth charts, and ability to use the Guidelines for Action.

Analytical Methodology

The total scores to the final tests as well as their different components were compared using t-tests, and confidence intervals were calculated using standard techniques.[8] The multivariate analysis of variance procedure (MANOVA)[9] was used to test main effects (chart, teaching period, training center) and interactions (particularly the carry-over effect, or treatment by period interaction); to control for baseline scores, age calculation scores and inter-hospital variability (using a nested design); and to obtain adjusted means and standard errors. The data were coded, entered and analyzed in Lesotho, using the Lesotho Food and Nutrition Coordinating Office (FNCO) microcomputer facilities, and the SPSS/PC+ statistical package.

Results

The overall score to the baseline test on growth charts knowledge was 3.4 out of 10 possible points and did not differ between training centers. Although work experience varied between participants (from 0 to 48 weeks), their poor knowledge of growth charts suggests that they had never received proper training in either chart.

A positive carry-over effect was found in the ANOVA model (significant interaction between chart and teaching schedule), meaning that prior training in one chart facilitated the learning of the second chart. Due to these findings, comparisons between the charts are presented separately for first training and for second week training.

Table 1 shows the results of the tests after training for all participants grouped by hospital and training schedule. In spite of similar baseline results, some differences between training centers were observed in the final scores. On average, Hospital B had higher scores than the other training centers, for both final tests, while Hospital C had lower scores. To control for the hospital variability, further analyses were done by analysis of variance (MANOVA procedure[9]), using a nested effect model (hospital within training schedule group). The baseline scores were also included in the model as covariates to adjust the final scores. The results of this analysis are presented in Table 2, which shows a comparison of the RTH and GS scores (unadjusted and adjusted) obtained on first week training and upon second week training, for the four hospitals grouped by training schedule. On first week training, the difference between the charts was minimal. Upon second week training, however, the scores to the RTH test were 15–20 percent higher (adjusted and unadjusted means, respectively) than the scores to the GS chart. This large difference between the charts resulted from a positive carry-over effect from first to second week training, observed only when the RTH chart was taught second: the GS-RTH group improved their score markedly from the first to the second week of training (+5.82, unadjusted means), while the RTH-GS group decreased their score from the first to the second week of training (−.76, unadjusted means). Examination of the components making up the total score to the final tests reveals that most of the carry-over effect seen in the GS-RTH group was due to the marked improvements in the interpretation and action questions (Table 1, Hospitals A and B).

TABLE 1—Results of the Final GS and RTH Tests, by Hospital and Training Schedule[a] (Group)

	GS Test				RTH Test			
Group/Hospital	Age[b]	Plotting[c]	Interpretation/ Action[d]	Total	Age	Plotting	Interpretation/ Action	Total
Group 1: GS-RTH[e]								
Hospital A	4.54	3.45	25.15	33.16	4.46	4.46	31.92	40.84
	(1.13)	(1.27)	(8.63)	(9.67)	(1.39)	(0.88)	(5.63)	(6.52)
Hospital B	4.00	4.00	33.90	41.90	4.09	4.38	38.09	46.57
	(0.95)	(1.00)	(6.47)	(7.30)	(1.73)	(1.28)	(3.05)	(4.33)
A + B	4.20	3.79	30.56	38.56	4.23	4.41	35.73	44.38
Pooled	(1.04)	(1.12)	(8.43)	(9.22)	(1.60)	(1.13)	(5.14)	(5.90)
Group 2: RTH-GS[e]								
Hospital C	3.10	2.30	23.50	28.90	3.50	3.20	24.00	30.70
	(1.52)	(1.70)	(8.42)	(11.03)	(0.97)	(1.50)	(8.31)	(9.47)
Hospital D	3.67	2.80	30.20	36.67	3.73	4.06	28.93	36.72
	(1.59)	(1.57)	(9.06)	(11.07)	(1.62)	(1.28)	(8.80)	(10.38)
C + D	3.44	2.60	27.52	33.56	3.67	3.72	26.96	34.42
Pooled	(1.56)	(1.61)	(9.26)	(11.50)	(1.38)	(1.43)	(8.79)	(11.50)

[a] The sample sizes were: Hospital A (n = 13), Hospital B (n = 21), Hospital C (n = 10), Hospital D (n = 15).
[b] Age: Calculation of age (maximum score = 5 points). Method was the same for both charts.
[c] Plot: Plotting of weights on charts (maximum score = 5 points).
[d] Interpretation and action: Interpretation of growth patterns and decision about actions to be taken, using the chart-specific Tables of Action (maximum score = 41).
[e] GS-RTH: GS taught first; RTH-GS: RTH taught first.
NOTE: The numbers in this table are means with standard deviations in parentheses.

Age calculation scores, on the other hand, did not improve from first to second training, but differed between hospitals, following a similar pattern as that observed for the overall scores. Since age calculation was independent of the charts, the differences observed between hospitals reflected variations in intrinsic learning capabilities of the health workers. An analysis of the sum of the interpretation and action scores was done that controlled for both hospital variability (using the nested design described previously) and differences in learning capability as proxied by age calculation confirmed the inferences drawn from Table 2. There was no substantial difference between the scores of the RTH and GS charts, −2.17 (with 95% confidence intervals (CI) of −6.67 to 2.33) after the first week of training. A significant improvement in the adjusted interpretation and action score from 31.81 ± 1.28 (standard error) to 38.16 ± 1.11 occurred when the RTH was taught in the second week. This contrasted with no improvement (from 33.98 ± 1.84 to 33.86 ± 1.59) when GS was taught in the second week. Thus the final adjusted score for interpretation and action was 4.3 (with CI of .43 to 8.17) in favor of RTH after the second week of training.

Discussion

In contrast to a previous study that tested health workers' skills in using different growth charts,[10] our study standardized the educational input across charts, and the participants were tested not only on their ability to understand, use, and interpret growth charts but also on their skills in using the Guidelines for Action to make the right decisions about a particular child. These guidelines were developed by Basotho experts involved and experienced in growth monitoring and maternal and child care. The feasibility in actual field conditions of implementing the actions proposed was an important consideration in the creation of these guidelines.

The present study had two major findings. First, at the end of the first train-

TABLE 2—Mean Scores to the GS and RTH Tests, by Group (Schedule of Training) and Week of Training[a]

	Results to the Final Test on		Difference between Charts
	GS Chart	RTH Chart	Difference (95% CI[b])
First week training			
	Group 1: GS-RTH (Hospitals A and B)	*Group 2: RTH-GS* (Hospitals C and D)	
Unadjusted means	38.56	34.32	+4.24 (−.84,9.32)
(SE)	(1.58)	(2.06)	
Adjusted means[c]	37.41	35.57	+1.84 (−3.94,7.63)
(SE)	(1.67)	(2.36)	
Second week training			
	Group 2: RTH-GS (Hospitals C and D)	*Group 1: GS-RTH* (Hospitals A and B)	
Unadjusted means	33.56	44.38	−10.82 (−5.90,−15.74)
(SE)	(2.30)	(1.01)	
Adjusted means[c]	36.04	43.77	−7.73 (−2.46,−13.00)
(SE)	(2.20)	(1.55)	

[a] Sample sizes were: group 1 (n = 34) and group 2 (n = 25).
[b] 95% confidence interval.
[c] The means were adjusted for hospital variability and for baseline scores, using the MANOVA[9] procedure and a nested design model (hospital within group).

ing week, the health workers who were taught the RTH understood their chart as well as those who were taught the GS chart. Thus, the argument that the GS was easier to learn for inexperienced African health workers was not substantiated. Second, when the participants were taught a second chart, those taught the RTH chart scored approximately 15 percent higher than those who were taught the GS chart. This improvement was not observed when the schedule of teaching was reversed: RTH first and GS second.

The improvement when RTH was taught second was achieved mainly through higher scores in the interpretation and action questions which measured the health workers' ability to identify appropriate interventions to be given to hypothetical children from the case histories. Since the guidelines were almost identical for the two charts,[*] it was expected that participants would master them better at the end of the second week, irrespective of the teaching sequence. However, our results showed no improvement on these questions and in the total score for those taught the GS chart on the second week (RTH-GS group). This finding suggests that prior knowledge of the RTH did not facilitate the learning of the GS chart and may even have confused the trainees because the visual presentation of growth in the GS chart is counterintuitive.

This finding is important since it is related to a situation that exists in many African countries: health workers that have been using a RTH-type chart for many years have to change to the GS system when the Catholic Relief Service Food and Nutrition Program is introduced; this requires intensive retraining of all the health workers. If the prior knowledge and experience with another chart (in this case the RTH) did facilitate the learning of the GS chart, the whole effort involved in the change of system would be reduced and potentially justifiable. However, the results of our study suggest that this was not the case in Lesotho.

Therefore, our conclusion from this study is that, in Lesotho, the RTH would be a better choice than the GS chart for two reasons. First, since health workers could learn and understand the RTH as well as the GS chart after the first week of teaching, it would be an appropriate chart for training inexperienced people. Second, and more important, in the context of Lesotho where retraining is a crucial issue, the RTH chart would be superior since second week training on the RTH improved the scores of those previously trained in the GS chart.

These recommendations are in accordance with those made in a concurrent study[5] showing that mothers who were taught the RTH learned significantly more about their chart than those who were taught the GS chart. Thus our overall conclusion from these two studies is that, in Lesotho, the RTH would be a more appropriate national chart than the GS. Nevertheless, it would be useful to replicate these results elsewhere in Africa where literacy of mothers and health workers is lower and more typical of much of Sub-saharan Africa. It would also be interesting to re-assess health workers' ability to use and interpret the two charts after use in growth monitoring activities.

We believe that the use of information from applied research, such as this, is fostered by the involvement of the national decision makers at all stages of the research process. In Lesotho, the decision was made to use a RTH-type chart on a nationwide basis, as recommended by our study. ☐

Acknowledgments

This research was funded by the Joint Program between UNICEF and Cornell Nutritional Surveillance Program, Cooperative Agreement # AID DSAN CA-0240 between USAID and Cornell University. Additional funding was provided by UNICEF. The authors wish to thank: Victoria J. Quinn from the Joint Program for her crucial work in the initiation and implementation of this research; John Haaga formerly from CNSP, now at the Rand Corporation, for writing the first research proposal; Martin Mogwanja, Assistant UNICEF Representative for Lesotho, for his remarkable efficiency and his continued support throughout the field work and at all stages of the research.

References

1. Grant J: Going for growth. *In*: State of the World's Children. Oxford: UNICEF and Oxford University Press, 1987; 64–80.
2. Rohde JE: Beyond survival: Promoting healthy growth. Ind J Pediatr 1988: 55(Suppl):S3–S8.
3. Griffiths M: Growth monitoring - making it a tool for education. Ind J Pediatr 1988; 55(Suppl):S59–S66.
4. Gopalan C: Growth monitoring - some basic issues. Bull Nutrition Foundation of India 1987.
5. Ruel MT, Pelletier DL, Habicht JP, Mason JB, Chobokoane CS, Maruping AP: A comparison of mothers' understanding of two growth charts in Lesotho. WHO Bulletin, 1990; 68:483–491.
6. World Health Organization: A growth Chart for International Use in Maternal and Child Health Care. Guidelines for Primary Health Care Personnel. Geneva: WHO, 1978.
7. Capone C: Integrating Title II Programs with Locally Operated Nutrition, Socio-economic, and Humanitarian Activities. Field Bulletin No 27, Nairobi, CRS, 1977.
8. Snedecor GW, Cochran WG: Statistical Methods, Seventh Ed. Ames: Iowa State University Press, 1980.
9. SPSS Inc, Norusis MJ: Advanced Statistics. SPSS/PC+ for the IBM PC/XT/AT. Chicago, IL: SPSS Inc, 1986.
10. Lacuna M: A Comparison of Three Growth Charts Used in Nutrition Programs by Field Nutrition Workers of Catholic Relief Services -USCC in Metro Manila. Manila: University of the Phillippines, 1979 (Master's Thesis).
11. Nabarro D, McNab S: A simple new technique for identifying thin children (The weight-for-height wall chart). Trop Med Hyg 1980; 83:21–23.

APPENDIX I

List of Institutions Participating in the Steering Committee in Charge of the Study

Name of Institution

Bureau of Statistics of Lesotho
Catholic Relief Services
Food and Nutrition Coordinating Office
Joint Program in Nutritional Surveillance of the UNICEF (Eastern and Southern Africa Regional Office) and the Cornell Nutritional Surveillance Program, Cornell University
Ministry of Agriculture of Lesotho
Ministry of Health of Lesotho
Private Health Association of Lesotho
Red Cross
UNICEF, Lesotho Country Program
World Health Organization

---

[*] The only difference was that for the RTH chart, some recommendations were made for children with constant weight (Appendix II). For the GS chart this was not possible because constant weight cannot be observed directly on the chart.

APPENDIX IIA— Guidelines for Action Based on the Interpretation of Growth Patterns Plotted on the Road-To-Health (RTH) or the Growth Surveillance (GS) Chart

Part 1. *For Children Re-attending the Clinic*

	Age 0–24 months		Age ≥25 months		
Direction of Growth	Gaining Weight	Constant/ Losing Weight	Gaining Weight	Constant Weight (only for RTH[a])	Losing Weight
Position of Dot					
A) Above 80%	• Regular plan of action[b] • Praise/ encourage	• Refer to nursing sister	• Praise/ encourage	• Regular plan of action (if constant weight for 3 months: refer to nursing sister	• Refer to nursing sister
B) Below 80%	• Regular plan of action • Home visit	• Refer to nursing sister • Home visit	• Encourage • Follow-up monthly	• Regular plan of action (if constant weight for 3 months: refer to nursing sister	• Refer to nursing sister

Part 2. *For New or Irregular Attenders*

	Age 0–24 months	Age ≥25 months
Position of Dot		
A) Above 80%	• Use "thinness chart"[c]	• Encourage monthly visits
B) Below 80%	• Use "thinness chart"	• Use "thinness chart"

Results of "Thinness Chart"

Green	Yellow	Red
• Praise/encourage • Encourage monthly attendance	• Refer to nursing sister	• Refer to nursing sister (very critical in children <24 months)

[a] On the GS chart, constant weight is not detected until the child's % weight-for-height (WTHT) drops. In this case, constant weight is detected on the GS chart by a downward slope, similar to the effect of a weight loss.

[b] See Appendix IIB.

[c] The "thinness chart" is a weight-for-height wall chart.[11] The interpretation of the color code is:
Green: WTHT between 70% and 80% of the NCHS standards
Yellow: WTHT between 80% and 90% of the NCHS standards
Red: WTHT between 90% and 110% of the NCHS standards.

APPENDIX IIB

Description of the Regular Plan of Action

REGULAR PLAN OF ACTION

1. Inform mother about her own child's growth (as seen on the growth chart). This should be done with all mothers.
2. Briefly interview mothers about their feeding practices. (Consider each age group: 0–3 months; 4–9 months; 9–12 months; over 1 year).
3. Give advice to mothers concerning specific feeding practices for each age group (taking mothers' reported practices into consideration). Suggested advice for each age group:

0–3 months
- Breastfeeding
- Introduce liquids

4–9 months
- Breastfeeding
- Introduce soft foods: cereals, fruits, vegetables, mashed/ pureed eggs

9–12 months
- Breastfeeding
- Continue soft foods
- Introduce solids
- Start meat/fish/ chicken/peas/ beans
- Give frequent meals (>4/day)

>12 months
- Breastfeeding
- Adult foods: 3 food groups
- Frequent meals (>4/day)

Preventing
AIDS

Evaluating AIDS Education Efforts

Preventing AIDS: A Guide to Effective Education for the Prevention of HIV Infection

By Nicholas Freudenberg, DrPH

236 pages, softcover, 1989

Preventing Aids sythesizes the collective experience of more than 130 AIDS educators nationwide and builds on case histories of AIDS education efforts, reviewing specific strategies used to contain the disease and providing a framework for decisions about future AIDS education activities.

Preventing AIDS will help you understand the social and political forces affecting AIDS educators. You'll also get practical advice on assessing a population's needs, developing educational materials, building coalitions, evaluating program effectiveness, and meeting the challenges of working with such specific populations as gay and bisexual males, IV drug users, youth, women, and the homeless.

An essential resource for health educators and administrators, nurses, teachers, activists, health department workers—anyone interested in AIDS education.

ORDER TODAY!

APHA Publication Sales
1015 15th Street, NW
Washington, DC 20005

APHA

Stock No. 050	$19.95	APHA Members (limit 2 copies);
	$28.50	Nonmembers

Postage and handling: U.S. $2.00 per book (up to 5 books); Foreign $5.00 per book (up to 3 books). All orders must be prepaid. Foreign orders must be paid with check drawn on U.S. banks. Do not send cash. Call (202) 789-5667 for volume discounts.

Charge by phone (minimum order $25.00). Call (202) 789-5636 to charge to VISA or MASTERCARD.

Public Health Briefs

Evaluating Needle Exchange: Do Distributed Needles Come Back?

Joseph Guydish, PhD, MPH, George Clark, Delia Garcia, Moher Downing, Patricia Case, and James L. Sorensen, PhD

ABSTRACT

We employed capture-recapture methods as a strategy for evaluating needle exchange. Needles distributed by the exchange at two time periods were marked with color coded bands indicating the date and site of distribution. Half of the marked needles (2,068/4,239) returned within two weeks of distribution, and 61 percent (2,593/4,239) returned during the study period. The rate of return for stationary exchange sites (63 percent) was greater than that for roving/mobile sites (51 percent; $\chi^2 = 28.6$, $p < .001$). Of all needles returned, 87 percent (2,248/2,593) returned to the site of original distribution. (*Am J Public Health* 1991;81:617–619)

Introduction

Needle exchange programs can potentially slow the spread of the human immunodeficiency virus (HIV) among injection drug users (IDUs), their sexual partners, and their children. Needle exchange is controversial, however, in that it contradicts traditional drug treatment philosophy and removes at least one barrier to drug use. Reports from programs abroad suggest that needle exchange does not increase drug use or needle use, or encourage individuals to begin using nedles.[1–5] Whether these programs slow the spread of HIV remains unanswered. United States needle exchange programs now operate in several urban areas,[6–8] and there is a pressing need for evaluation. Strategies that do not rely on self-report can assist efforts to determine program effects.

One question is whether distributed needles return to the exchange. In the ideal program 100 percent of needles distributed would be returned in a short time, implying consistent use of new needles and a low likelihood of needle sharing in the exchange population. We employed capture-recapture methods to determine what proportion of distributed needles return to the exchange, how quickly needles return, and whether distributed needles travel between exchange sites.

The San Francisco exchange program (Prevention Point) trades needles on a 1:1 basis during regular hours on one night each week, and any patron may exchange up to 10 needles. There are two "roving" teams which travel on foot over a prescribed route, and two stationary teams. Together these sites exchanged an average of 2,806 needles per week during the study period.

Methods

Needles were identified for study purposes ("captured") with colored bands painted onto the barrel of each syringe. The color indicated both the site and date of distribution. Marked needles were distributed on two occasions. At Time 1, all needles distributed at Stationary Site A (n = 1,200) and Roving Site A (n = 204) were marked. At Time 2, nine weeks after the first distribution, marked needles were distributed at Stationary Site A (n = 1,300), Stationary Site B (n = 1,137), Roving Site A (n = 249), and Roving Site B (n = 149). These needles, marked with four colors different from those used at Time 1, comprised 95 percent (2,835/2,978) of all needles distributed at Time 2. After each distribution, all needles returned to any exchange site were inspected for a nine-week period, and banded needles (those "recaptured") were recorded.

This method is observational and unobtrusive. Exchange patrons were not interviewed, and no attempt was made to determine whether the original recipient of marked needles was also the person who returned them. Only needles collected by the exchange program were monitored, so no data are available concerning marked needles that did not return to the program. Prevention Point had distributed marked needles in conjunction

---

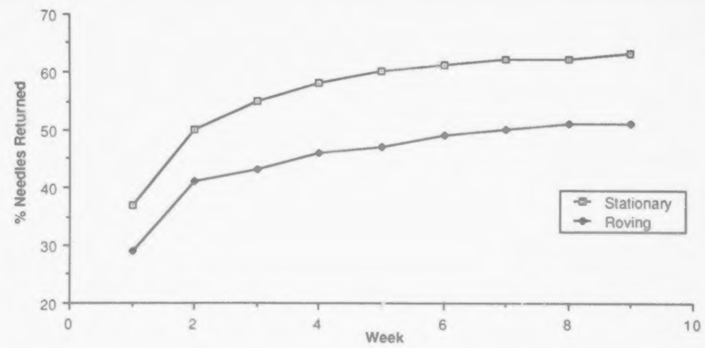
From the Center for AIDS Prevention Studies, University of California, San Francisco (Guydish, Sorensen) and the Prevention Point Research Group, San Francisco (all other authors). Address reprint requests to Joseph Guydish, PhD, Center for AIDS Prevention Studies, 74 New Montgomery Street, Suite 600, San Francisco, CA 94105. This paper, submitted to the Journal July 12, 1990, was revised and accepted for publication January 8, 1991.

TABLE 1—Rate of Return for Banded Needles, Collapsed across Time of Distribution and Site

Week of Return	Needles Returned N = 4,239* (%)
1	1500 (35.4**)
2	568 (13.4)
3	195 (4.6)
4	129 (3.0)
5	83 (2.0)
6	37 (0.9)
7	30 (0.7)
8	34 (0.8)
9	17 (0.4)
Totals	2593 (61.2)

*Total number of banded needles distributed.

**Percentages rounded to one decimal place.



Proportion returned for Stationary Sites is 62.8% (2284/3637), and for Roving Sites, 51.3% (309/602). $X^2 = 28.6$, $p < .001$

FIGURE 1—Cumulative Rate of Return by Type of Exchange Site, Collapsed across Time Periods

with a previous study,[6] so that many exchange patrons were accustomed to receiving marked needles. In the present study, patrons who asked about the markings were told that we wanted to learn how long distributed needles remained on the street.

Results

Results are presented for Time 1 and Time 2 combined, since the proportion of needles returned (60 percent and 62 percent, respectively) and the general pattern of return were the same. The rate of return was inversely related to time since distribution, with the greatest return occurring in the first five weeks (Table 1).

For the stationary site evaluated at both time periods, there was no difference in rate of return across time (63 percent vs 62.9 percent). For the roving site, the rate of return was greater at Time 2 than at Time 1 (58.2 percent vs 43.1 percent; $\chi^2 = 10.2$, $p < .01$). At Time 2 there were no differences in rate of return for the two stationary sites (62.9 percent vs 62.4 percent), or the two roving sites (58.2 percent vs 51 percent). To evaluate differences in return rate by type of site, we collapsed data across time for stationary and roving sites (Figure 1).

Last, we evaluated how often needles returned to the site where they were distributed. Of the banded needles returned, 87 percent (2,248/2,593) were returned to the site of original distribution. For stationary exchanges the proportion of needles returning to the home site ranged from 88 percent to 94 percent. The proportion returning home to Roving Site A was lower (74 percent). Unexpectedly, only 12 percent (9/76) of returning Roving Site B needles came back to the home site, and 82 percent returned to Stationary Site B located in the same neighborhood.

Discussion

For both Time 1 and Time 2, nearly 50 percent of distributed needles returned within two weeks. This finding suggests some stability in the exchange population, and that many patrons use the exchange repeatedly. The difference observed for rate of return between stationary and roving sites suggests that these models, in San Francisco, may serve different IDU populations. Roving teams were designed to reach homeless IDUs who are more transient and whose needles are frequently lost or stolen. To the degree that roving teams reach this population, a lower rate of return would be expected. The finding that needles distributed by Roving Site B frequently returned to Stationary Site B indicates that these sites serve an overlapping population.

The application of nonself-report methods is vital in adequately evaluating the effects of US needle exchange programs. While such methods cannot replace self-report strategies, they can provide independent verification of self-report data. The capture-recapture method indicates the stability of the exchange population, the frequency with which patrons use the exchange service, and the degree to which different exchange sites serve unique populations. ☐

Acknowledgments

The authors gratefully acknowledge the assistance of members of the volunteer community based San Francisco needle exchange, Prevention Point, and the needle exchange patrons. George Chu, PhD, made early conceptual contributions to the project, and Norman Hearst, MD, contributed substantively to preparation of the manuscript.

Some of the data included in this manuscript were presented at the VI International Conference of AIDS, San Francisco, June 1990 (Abstract S.C.762)

This work was supported by the AIDS Clinical Research Center, University of California, San Francisco.

References

1. Buning EC, van Brussel GHA, van Santen G: Amsterdam's drug policy and its implications for controlling needle sharing. *In*: Battjes RJ, Pickens RW (eds): Needle Sharing among Intravenous Drug Abusers: National and International Perspectives. Rockville, MD: National Institute on Drug Abuse, NIDA Research Monograph Series 1988; 59–74.
2. Hartgers C, Buning EC, van Santen GW, Verster AD, Coutinho RA: The impact of the needle and syringe-exchange programme in Amsterdam on injecting risk behavior. AIDS 1989; 3:571–576.
3. van den Hoek JAR, van Haastrecht HJA, Coutinho RA: Risk reduction among intravenous drug users in Amsterdam under the influence of AIDS. Am J Public Health 1989; 79:1355–1357.
4. Stimson G: Injecting equipment exchange schemes: Final report. Monitoring Research Group, Goldsmith's College, New Cross, London SE14 6NW, UK. November 1988.
5. Wolk J, Wodak A, Morlet A, *et al*: Syringe

HIV seroprevalence and behavioural and demographic characteristics of intravenous drug users in Sydney, Australia, 1987. AIDS 1988; 2:373–377.

6. Clark GL, Downing M, McQuie H, *et al*: Street-based needle exchange programs: The next step in HIV prevention. Poster presented at the V International Conference on AIDS, Montreal, June 1989 (Abstract Th.D.P.73).
7. Purchase D, Hagan H, Des Jarlais DC, Reid T: Historical account of the Tacoma syringe exchange. Poster presented at the V International Conference on AIDS, Montreal, June 1989 (Abstract Th.D.P.74).
8. New York City Department of Public Health: The pilot needle exchange study in New York City: A bridge to treatment. New York City Department of Public Health, 125 Worth Street, New York, NY, December 1989.

Targeted HIV Screening at a Los Angeles Prenatal/Family Planning Health Center

Laura J. Fehrs, MD, Deirdre Hill, MPH, Peter R. Kerndt, MD, Thanne P. Rose, PhD, and Carol Henneman, MD

ABSTRACT

In 1988, the Los Angeles County Health Department conducted a blinded human immunodeficiency virus (HIV) seroprevalence study at a public prenatal (PN) and family planning (FP) center serving mostly Hispanic women in order to determine seroprevalence and to evaluate the center's targeted HIV screening program. Four women (0.13 percent) tested positive (3/1801 PN and 1/1167 FP). Three reported no risk factors; one reported a history of syphilis since 1978. Voluntary HIV testing was selectively offered to women who reported risk factors for HIV infection. Only 14 percent (96/685) of clients offered testing chose to do it: 28 percent (14/50) of clients classified as being at highest risk of infection, and 27 percent (16/59) of women who judged themselves to have some chance of being exposed to HIV. None of the four women who tested positive by blinded testing chose testing. While few women at this center were infected with HIV, higher risk women were not persuaded to be tested through a targeted screening program. Blinded HIV seroprevalence studies provide a tool for both tracking infection in a population and evaluating screening programs. (*Am J Public Health* 1991;81:619–622)

Introduction

Women of childbearing age in the United States are at risk for human immunodeficiency virus (HIV) infection although their risk varies by locality, risk group, and race.[1–14] In 1987, the US Public Health Service (PHS) recommended that women with identifiable risks for HIV infection be routinely counseled and tested for HIV antibody.[15] Strategies for providing women of childbearing age with HIV counseling and testing include: 1) routine or universal screening, with informed consent, of women attending women's health clinics, such as prenatal (PN) or family planning (FP) clinics and 2) selective targeting of women identified as having risk factors for HIV infection. In the latter approach, risk factors for HIV infection are assessed during a counseling session and those patients reporting risk factors are offered voluntary testing. In areas with high rates of HIV seropositivity among women, this approach has not been shown to be effective in identifying most seropositive women.[3,4,8,10,16] Fewer data exist for areas with low or unknown rates of seropositivity among women, such as Los Angeles.

In 1988, the Los Angeles County Department of Health Services participated in the PHS Family of Surveys[17] and initiated a blinded HIV seroprevalence study at a large inner-city public health center which used a targeted approach for HIV counseling and testing in its PN and FP clinics. Results of blinded HIV testing were used to evaluate the extent to which the HIV counseling and testing program reached women who were either at risk for HIV infection or already infected.

Methods

Consecutive sera remaining after routine syphilis testing during initial or annual visits of all women attending the PN clinic from March through September, and the FP clinic from March through November, 1988 (3,000 total) were blinded by the removal of personal identifiers and then tested for HIV antibody by a licensed enzyme linked immunosorbent assay (ELISA) kit. Repeatedly reactive specimens were confirmed with immunofluorescent antibody (IFA) (Laboratory Services Branch, California State Department of Health Services) and Western blot.

The clinics used a targeted HIV counseling and testing program. All patients were counseled by trained bilingual interviewers about risk factors for HIV infection and the availability of HIV testing. The interviewers used a questionnaire

---

Address reprint requests to Laura J. Fehrs, MD, Division of Immunization, Centers for Disease Control, Atlanta, GA 30333; Drs. Kerndt and Fehrs were assigned to the Los Angeles County Department of Health Services from the CDC Epidemiology Program Office at the time of the study. Ms. Hill and Dr. Kerndt are with the AIDS Epidemiology Office; Dr. Henneman is with the H. Claude Hudson Comprehensive Health Center. All are with the LA County Department of Health Services. Dr. Rose was with the AIDS Epidemiology Office at the time of the study. She is currently with the Academy of Pediatrics in Chicago, Ill. This paper, submitted to the Journal May 22, 1990, was revised and accepted for publication November 20, 1990.

TABLE 1—Definition of Risk for HIV Infection Used by Clinic Staff and by Investigators, and Prevalence of Risk Behaviors, Los Angeles, 1987–88

	Risk behaviors (+) for which clinic staff offered voluntary testing	Risk behaviors (+) for which women were classified as "high risk" by investigators	Women with risk behavior No. (%)
Intravenous (IV) Drug Use since 1978	+	+	8 (0.3)
Sex since 1978 with an IV drug user, bisexual man, or person with AIDS or HIV infection	+	+	47 (1.6)
Receipt of money or drugs for sex in the last year	+	+	12 (0.4)
Transfusion from 1978 through 1985	+		107 (3.6)
Sex since 1978 with a prison inmate	+		172 (5.7)
Sex since 1978 with a man with hemophilia	+		0 (0)
Sex since 1978 with a man from Haiti or Central Africa	+		6 (0.2)
History of hepatitis	+		48 (1.6)
History of a sexually transmitted disease since 1978	+		38 (1.3)
History of more than one sex partner in the last year	+		165 (5.5)
History of sexual assault	+		60 (2.0)

TABLE 2—Results of HIV Antibody Seroprevalence Study, Los Angeles, 1987–88

		HIV Antibody Seroprevalence		
	% of All Patients	No. HIV Positive	Total No. Tested	% Positive
Clinic Type				
Prenatal	60.0	3	1801	0.17
Family Planning	38.9	1	1167	0.09
Unknown	1.1	0	32	0
Risk For Infection				
No Risk Behaviors Reported	77.0	3	2311	0.13
"At Risk" (Any risk behaviors reported)	23.0	1	689	0.15
"High risk"*	1.7	0	50	0
Self-Assessed Risk of ≥ 1 in 100 chances**	2.0	0	60	0

*Subset of "At Risk" group; these two groups were not mutually exclusive. See Table 1 for definitions.

**Self-assessment of risk of exposure was determined by interviewer asking patient "How likely do you think it is that you have been exposed to someone infected with the AIDS virus in such a way that you could be infected?" Patients were given the choice of answering "Very unlikely (less than 1 in 100 chance)", "Unlikely (1 in 10 chance)", "Uncertain (50-50 chance)", or "Certain (sure that you have been exposed to someone infected with the AIDS virus)."

as a guide to counseling and as a record of risk behavior information. To identify all women possibly at risk, the clinic chose to use a broad range of behaviors in its definition of risk (Table 1) and patients were also asked to assess their own risk (Table 2). Voluntary HIV testing was recommended and immediately offered to all women who requested testing and to those reporting any of the behaviors defined by the clinic to indicate possible risk.

The targeted HIV counseling and testing program was evaluated by using a unique study number to link the results of blinded seroprevalence testing to questionnaire information. Women were classified by their answers as being "at risk," "at high risk," or "not at risk." The "at risk" group was composed of those women who reported any of the broad range of risk behaviors which the clinic considered to place women at some risk of infection (the group that was offered HIV testing). A subset of "at risk" women, "high-risk" women, was defined by investigators to be at particularly high risk for HIV infection, using objective criteria (Table 1).

Confidence limits for proportions were calculated using the method published by Fleiss.[18]

Results

A total of 1,801 consecutive specimens from the PN clinic, 1,167 consecutive specimens from the FP clinic, and 32 specimens for which clinic type was not known were tested for HIV antibody (Table 2). Of the patients for whom race was known (2,955), 87.3 percent (2,579) were Hispanic, 9.6 percent (285) White, 2.4 percent (71) Black, 0.5 percent (15) Asian, and 0.2 percent (5/2,955) other race. The median reported length of residence in the US was five years. The median age was 25 years with a range of 12–46 years; age was unknown for 29 women.

Four of the 3,000 specimens (0.13 percent, 95 percent confidence interval (CI) = 0.04, 0.37) were repeatedly reactive by ELISA and positive by IFA and Western Blot; three were from the PN clinic (0.17 percent, 95% CI = 0.04, 0.55), and one was from the FP clinic (0.09 percent, 95% CI = 0.00, 0.55) (Table 2). These positive specimens were from one White and three Hispanic patients, all of whom were in the 20–24 age group. The length of time that these patients reported residing in the US ranged from two to 12 years.

None of the four seropositive women chose voluntary HIV testing (Table 3). Only one of the four reported any risk factor for infection—a history of syphilis since 1978 (Table 2). Because this finding was of concern, we further investigated the center's testing strategy by determining the rate of acceptance of testing among those reporting risk behaviors.

While 23 percent of women reported one or more risk behaviors (were considered "at risk" by clinic staff) and 1.7 percent met the criteria for classification as "high risk," only 2.0 percent judged themselves to have more than a one in 100 chance of being exposed to HIV infection (Table 2). A high proportion of women who were "at risk" (92 percent) or even "high risk" (66 percent) assessed their own risk for being exposed to HIV infection to be "very unlikely" (less than a one in 100 chance) (Table 4). All four of the

TABLE 3—Acceptance of Voluntary HIV Testing, by Risk Group

Risk Group	No. Accepted Voluntary HIV Testing	Total No. in Group*	% Accepted Voluntary Testing
Reported Risk Behaviors			
"At Risk" (Any risk behaviors reported)	96	685	14.0
"High risk"	14	50	28.0
Self-Assessed Risk	16	59	27.1
Tested Positive in HIV Seroprevalence Study	0	4	0
Tested Negative in HIV Seroprevalence Study	110	2985	3.7

*Persons for whom acceptance of testing was not known were excluded from analysis

seropositive women also assessed their own risk as being "very unlikely."

Not only did the four seropositive women refuse HIV testing, but only 14.0 percent of women offered testing because of a history of risk behaviors ("at risk" group) consented (Table 3). Among the women who were classified as being "high risk" and among the women who judged their chance of exposure to be greater than one in 100, only 28.0 percent and 27.1 percent, respectively, selected voluntary testing.

Comments

The prevalence of HIV antibody among prenatal and family planning patients attending this inner-city, mostly Hispanic, public health center in Los Angeles was low—0.13 percent (95% CI = 0.04, 0.37). The rate found in this study is consistent with the low prevalence of antibody reported from parturients (0.04 percent),[19] newborn screening (0.10 percent),[20] and FP clinics (0.1 percent, D. Hill, unpublished data) in Los Angeles County. Since the population in this study was predominantly Hispanic and immigrant, it should not be considered representative of all clinics with a high proportion of minority clients. But, as one of six large comprehensive public health centers in Los Angeles, it is an important site for monitoring HIV infection.

While the seroprevalence found at this center was reassuringly low for an urban area with a high incidence of AIDS, it is of concern that the program of targeted HIV screening did not identify the seropositive women in this study. Three of these four women reported no risk factors for infection. The rates of reporting of risk behaviors and acceptance of testing in this population may have been affected by cultural factors and/or lack of knowledge of risk behaviors of partners. Some women may have been tested already for HIV infection for immigration or other purposes. Acceptance of testing may have been affected in unknown ways by the counseling process; however, in this regard, the clinic is probably typical of urban, high-volume clinics.

Our data suggest that a strategy of selectively offering HIV voluntary testing only to those women who report risk factors for infection may result in a low rate of acceptance and may miss seropositive women. Similar data from several studies led the US Public Health Service's Immunization Practices Advisory Committee to recommend the universal screening of PN patients for hepatitis B.[21–26] Universal screening for HIV of all women pregnant or contemplating pregnancy has been recommended[1–4,16,27,28] because it identifies a high proportion of infected women; these women can be referred early to medical prophylaxis and treatment; and it results in the early identification and treatment of children born to HIV-infected mothers. Universal screening, with informed consent, has been conducted at some Los Angeles prenatal clinics since 1988.

Despite the benefits of universal screening, targeted screening may be used in localities with a low prevalence of HIV or without resources for adding large scale HIV testing and counseling to routine clinical practice. Efforts to identify seropositive women compete for limited resources with HIV prevention and other public health programs. In a 1988–89 random survey of urban hospitals, most did not offer HIV testing routinely to pregnant women.[28]

Those who design voluntary HIV screening programs for women should consider: local seroprevalence; available resources vs demand for testing and other services; local characteristics of women at risk for infection; strategies that result in high levels of acceptance of testing among women at risk; and strategies that provide counseling to seronegative women and effect behavioral changes that will prevent the future transmission of HIV.

The variety of strategies for screening women of childbearing age should be evaluated locally and over time. Blinded HIV seroprevalence studies provide valuable data about voluntary screening programs. In addition, they supplement acquired immunodeficiency syndrome case

TABLE 4—Self-Assessment of Risk of Significant Exposure to HIV Infection, by Risk Group, Los Angeles, 1987–88

Self-assessment of Risk of Exposure	Risk Group: "High risk" No.	%	"At Risk" No.	%	No Risk Factors No.	%
Very unlikely (<1 in 100 chance)	33	66.0	633	91.9	2268	98.1
Unlikely (1 in 10 chance)	6	12.0	17	2.5	14	0.6
Uncertain (50-50 chance)	10	20.0	19	2.8	6	0.3
Certain (sure was exposed)	1	2.0	3	0.4	1	0.04
No response	0	0	17	2.5	22	0.7
Total	50	100	689	100	2311	100

surveillance by tracking trends in HIV infection among women over time. ☐

References

1. Novick LF, Berns D, Stricof R, Stevens R, Pass K, Wethers J: HIV seroprevalence in newborns in New York State. JAMA 1989; 261:1745–1750.
2. Donegan SP, Edelin KC, Craven DE: HIV seroprevalence rate at the Boston City Hospital. N Engl J Med 1988; 319:653–654.
3. Landesman S, Minkoff H, Holman S, *et al:* Serosurvey of human immunodeficiency virus infection in parturients. Implications for human immunodeficiency virus testing programs of pregnant women. JAMA 1987; 258:2701–2703.
4. Krasinski K, Borkowsky W, Bebenroth D, *et al:* Failure of voluntary testing for human immunodeficiency virus to identify infected parturient women in a high-risk population. N Engl J Med 1988; 318:185.
5. Altman R, Grant CM, Brandon D, *et al:* Statewide HIV-1 serologic survey of newborns with resultant changes in screening and delivery system policy. Abstract WAO7. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
6. Novick L, Glebatis D, Stricof R, *et al:* HIV infection in adolescent childbearing women. Abstract WAO8. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
7. Withum DG, LaLota M, Holtzman D, *et al:* Prevalence of HIV antibodies in childbearing women in Florida. Abstract MAP6. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
8. Barbacci M, Quinn T, Kline R, *et al:* Failure of targeted screening to identify HIV positive pregnant women. Abstract MBP10. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
9. Connor E, Danny T, Goode L, *et al:* Seroprevalence of HIV-1 and HTLV-I among pregnant women in Newark, NJ. Abstract MBP19. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
10. O'Sullivan MJ, Fajardo A, Ferron P, *et al:* Seroprevalence in a pregnant multi-ethnic population. Abstract MBP23. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
11. Duttel BJ, Hauer LB, Crombleholme W, *et al:* HIV-1 seroprevalence and risk behavior are increased in pregnant women receiving no prenatal care. Abstract MBP33. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
12. Killian H, Risi G, James D, *et al:* HIV infection rates in parturients who receive no prenatal care. Abstract B504. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
13. Newstetter A, Eversley RB, Biernes DL: Seroprevalence among a low HIV prevalence population. HIV screening data from a family planning population. Abstract D683. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
14. Crane M, Tortolero S, Garcia G, *et al:* Risk factors and HIV seroprevalence in female clients of selected health department clinics, Houston, Texas. Abstract E716. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
15. Centers for Disease Control: Public Health Service guidelines for counseling and antibody testing to prevent HIV infection and AIDS. MMWR 1987; 36:509–515.
16. Kaunitz AM, Brewer JL, Paryani SG, *et al:* Prenatal care and HIV screening. JAMA 1987; 258:2693–2694.
17. Pappaioanou M, Dondero TJ, Petersen LR, Onorato IM, Sanchez CD, Curran JW: The family of HIV seroprevalence surveys: Objectives, methods, and uses of sentinel surveillance for HIV in the United States. Public Health Rep 1990; 105:113–119.
18. Fleiss JL (ed): *Statistical Methods for Rates and Proportions*. New York: John Wiley & Sons; 1981; 14–17.
19. Hill D, Kerndt P, Frenkel LM, *et al:* HIV Seroprevalence among parturients in Los Angeles County, 1988. Abstract MAP12. Presented at the Fifth International Conference on AIDS, Montreal, Canada, June 4–9, 1989.
20. California Department of Health Services, Office of AIDS: State health director announces final results of study of HIV infection. News release 69-89, September 7, 1989, Sacramento.
21. Centers for Disease Control: Prevention of perinatal transmission of hepatitis B virus. Prenatal screening of all pregnant women for hepatitis B surface antigen. MMWR 1988; 37:341–351.
22. Jonas MM, Schiff ER, O'Sullivan MJ, *et al:* Failure of Centers for Disease Control criteria to identify hepatitis B infection in a large municipal obstetrical population. Ann Intern Med 1987; 107:335–337.
23. Kumar ML, Dawson NV, McCullough AJ, *et al:* Should all pregnant women be screened for hepatitis B? Ann Intern Med 1987; 107:273–277.
24. Malecki JM, Guarin O, Hulbert A, *et al:* Prevalence of hepatitis B surface antigen among women receiving prenatal care at the Palm Beach County Health Department. Am J Obstet Gynecol 1986; 154:625–626.
25. Delage G, Montplaisir S, Remy-Prince S, *et al:* Prevalence of hepatitis B virus infection in pregnant women in the Montreal area. Can Med Assoc J 1986; 134:897–901.
26. Wetzel AM, Kirz DS: Routine hepatitis screening in adolescent pregnancies. Is it cost effective? Am J Obstet Gynecol 1987; 156:166–169.
27. Rhame FS, Ma[illegible] DG: The case for wider use of testing for HIV infection. N Engl J Med 1989; 320:1248–1254.
28. Schwartz RM, Dresser M, Maynard EC: Hospital policy, practice re HIV testing for pregnant women and newborns. Am J Public Health 1990; 80:1276.

HIV-1 Prevalence in Selected Tijuana Sub-populations

ABSTRACT

To assess the prevalence of HIV-1 (human immunodeficiency virus) infection among high-risk populations in Tijuana, Mexico, HIV-1 antibody status was determined and information on risk behavior was obtained from 1,069 individuals in three high-risk groups. The prevalence of HIV-1 among 415 prostitutes was 0.5 percent; 410 prisoners, 1.2 percent; 233 homosexual/bisexual men, 11.6 percent; and 106 intravenous drug abusers, 1.9 percent. The potential for spread of HIV-1 exists in Tijuana despite the current relatively low seroprevalence of HIV-1. (*Am J Public Health* 1991;81:623–625)

Fernando Güereña-Burgueño, MD, Abram S. Benenson, MD, and Jaime Sepúlveda-Amor, MD

Introduction

By December 31, 1989, 3,512 AIDS (acquired immunodeficiency syndrome) cases have been reported to the Mexican Ministry of Health.[1] Projections made by the Mexican National Committee for AIDS Prevention (CONASIDA) suggest that the cumulative AIDS case total could exceed 75,000 by the end of 1991.[2] The majority of the 71 AIDS cases in the state of Baja California[2] have been reported by the city of Tijuana, geographically contiguous to San Diego, California. This border area is the busiest port of entry to the United States, with approximately 36 million legal entries of alien and US citizens, and more than 500,000 illegal aliens deported in 1987.

This study presents the results of a cross-sectional epidemiologic and serological survey designed to determine the prevalence of infection with the human immunodeficiency virus type 1 (HIV-1) among "high-risk" populations in Tijuana. The project was approved by the Committee on Protection of Human Subjects of the San Diego State University.

Methods

Sample sizes were selected to provide less than 3 percent margin of error ($\alpha = .05$ and $1-\beta = .80$) (4). From 1,967 prisoners in the Tijuana State Penitentiary, 410 were recruited by selecting every fifth prisoner listed in the prison records. From 2,000 active prostitutes registered at the Municipal Medical Services Clinic, 415 were sequentially included in the study. A convenience sample of 233 homosexual/bisexual men was recruited in diverse homosexual gathering sites. From 1,000 intravenous drug abusers with files at the Juvenile-Integration Center detoxification clinic at Tijuana, a sample size of 341 was included in the study by selecting every third individual listed in the clinic records.

Participation was voluntary and anonymous. Each subject was individually counseled prior to testing. Sociodemographic and risk factor information was obtained and a blood sample was drawn. Test results with post-test counseling were offered to the participants.

Antibodies against HIV-1 were determined with licensed enzyme-immunoassay (EIA) kits.[5] Those repeatedly reactive were then confirmed by the indirect immunofluorescent antibody assay (IFA)[6,7] or by the Western blot technique.[8]

Descriptive statistics and odds ratios were calculated and 95 percent confidence intervals estimated with Cornfield's method.[9]

Results

Questionnaires were completed and blood samples obtained from 1,069 individuals. The refusal rate was 2 percent in the prisoner and prostitute groups and 25 percent to 30 percent in the group of homosexual/bisexual men. Only 11 of the 341 drug abusers in the files of the detoxification clinic could be located. However, 95 individuals in the other three groups admitted intravenous drug abuse (IVDA).

A total of 34 individuals tested positive for HIV-1. The prevalence rates by study group, sex, and history of IVDA are shown in Table 1.

Seropositive homosexual/bisexual men exhibited high-risk behavior patterns (Table 2); a history of receptive anal sex showed the strongest relation to HIV-1 infection. For vaginal sex, 36 percent (139/383) of prostitutes reported using condoms "usually" or "always." Of female prostitutes and female prisoners participating in the study, 12.3 percent (46/383) and 5.9 percent (2/34), respectively, reported anal sex.

From the Graduate School of Public Health, San Diego State University California (Güereña-Burgueño and Benenson) and the General Directorate of Epidemiology, Ministry of Health, Mexico (Sepúlveda-Amor). Address reprint requests to Fernando Güereña-Burgueño, MD, Division of HIV/AIDS, Centers for Disease Control, 1600 Clifton Road, NE, Mailstop G-29, Atlanta, GA 30333. This paper, submitted to the Journal April 2, 1990, was revised and accepted for publication October 10, 1990.

TABLE 1—Prevalence of HIV-1 Antibody by Study Group, Tijuana HIV-1 Seroprevalence Study, June–October 1988

	Total		Intravenous Drug Abusers*		Non-intravenous Drug Abusers	
Study Group	No. Subjects	Prevalence (%)	No. Subjects	Prevalence (%)	No. Subjects	Prevalence (%)
Prostitutes						
Males	1/32	(3.1)	1/2	(50.0)	0/30	(0.0)
Females	1/383	(0.3)	0/3	(0.0)	1/380	(0.3)
Total	2/415	(0.5)	1/5	(20.0)	1/410	(0.2)
Prisoners						
Males	5/376	(1.3)	0/81	(0.0)	5/295	(1.7)
Females	0/34	(0.0)	0/4	(0.0)	0/30	(0.0)
Total	5/410	(1.2)	0/85	(0.0)	5/325	(1.5)
Homosexual/bisexual men	27/233	(11.6)	1/5	(20.0)	26/228	(11.4)
IVDA at the CIJ-clinic	0/11	(0.0)	0/11	(0.0)	0/0	(0.0)
Total	34/1069	(3.2)	2/106	(1.9)	32/963	(3.3)

*101 reported IV drug abuse since 1978

TABLE 2—Behavior Patterns among Homosexual/Bisexual Men by HIV-1 Antibody Status, Tijuana HIV-1 Seroprevalence Study, June–October 1988

	Seropositive		Seronegative			
Behavior	N	(%)	N	(%)	OR*	95% CI**
History of drug abuse	16/27	(59.3)	87/206	(42.3)	2.0	0.82, 4.9
History of sexually transmitted disease	15/27	(55.6)	83/206	(40.3)	1.9	0.77, 4.5
History of receptive anal sex	21/27	(77.8)	113/206	(54.9)	2.9	1.0, 8.4
History of prostitution	11/27	(40.7)	67/206	(35.5)	1.4	0.58, 3.5
History of sex in the US	12/27	(44.4)	60/206	(29.1)	2.0	0.80, 4.7
Usually or always using condoms	5/27	(18.5)	55/206	(26.7)	0.62	0.20, 1.9

*Odds ratio
**Comfield's 95% confidence limits

Within the penitentiary, 30 percent (117/410) of the prisoners reported current use of illegal drugs, 47 percent of the current users reported using IV drugs. Regular use of condoms was reported by only 4.2 percent (17/403) of the sexually active prisoners.

Discussion

This study is the largest survey reported of HIV-1 seroprevalence among potential high-risk populations in Tijuana. Nevertheless, it was impossible to reach and make precise estimates of the number and location of the homosexual/bisexual men; self-selection may have biased the seroprevalence estimated in this group. The main reason of failure to find the drug abusers selected from the clinic, was that most of them were residents of the neighboring state of California, who came to Tijuana for treatment. The almost complete lack of refusal among the prisoners and prostitutes groups actually eliminates concern of selection bias. However, bias could have been introduced when obtaining information by questionnaire regarding sensitive issues, such as sexual behavior and drug abuse.

Plausible hypotheses can be offered to explain the relatively low seroprevalence of HIV-1 infection found in this study. First, the number of partners claimed by the homosexual group is lower than that reported in high incidence places.[10–12] Second, the availability in pharmacies of sterile syringes without the requirement of a medical prescription may result in less sharing of used needles among drug abusers. However, the 1.9 percent prevalence of HIV-1 positivity in this group was similar to that estimated in various studies conducted in the Southern California area, ranging from 0 percent to 7 percent.[13] Third, the low rate among prostitutes may be attributed to the increased health consciousness of this group with monthly medical contact. Fourth, the legal weekly permission for the prisoners to receive conjugal visits by spouses or prostitutes might have lessened the possibility of widespread unprotected homosexual activity among the prisoners; however, 5 percent (19/376) of the prisoners reported history of homosexual contact during the last year. The lower availability of sterile syringes in this site and the low use of condoms indicate a serious potential of spread of HIV-1 should the virus be brought into the prisoner group.

Experience has shown that reduction in HIV-1 transmission, as well as other sexually transmitted diseases, is possible when reduction of high-risk sexual practices is induced by means of preventive intervention.[14–16] Logistic support in the implementation of preventive efforts needs to be permanently offered to nongovernmental AIDS organizations by the health authorities and coordinated and complementary action by the two countries must follow. ☐

Acknowledgments

The collection of data herein described was a collaborative effort of the Graduate School of Public Health, San Diego State University, Division of Epidemiology and Biostatistics; the General Directorate of Epidemiology, Ministry of Health, Mexico; and the State of California Department of Health and Human Services. The authors wish to thank Adela Caudillo, MD, and Jesús Bucardo, MD, the Graduate School of Public Health, San Diego State University; Kenneth W. Kizer, MD, Director of the State of California Department of Health Services; Michael S. Ascher, MD, Deputy Chief, and Duc J. Vugia, MD, State of California Viral and Rickettsial Disease Laboratory; Mr. Emilio Velázquez, Organización SIDA Tijuana; Manuel Monlina Bellini, MD, and J. Jesús Curiel, MD, the Juvenile-Integration Center (CIJ); Gloria Salazar, RN, Director of the UABC-IMSS School of Nursing; Francisco Díaz Martínez, MD, Director of the Municipal Medical Services; Mr. Ernesto Moreno Montiel, Director of the Tijuana State Penitentiary; Remedios Lozada, MD, from the Sanitary Jurisdiction No. 2 SSA, and to all the volunteers for their cooperation. This study was supported by the State of California Department of Health and Human Services and the Graduate School of Public Health, San Diego State University.

This paper was presented at the National Conference on AIDS, Mexico City, Mexico, November 13–16, 1989, and at the 118th Annual meeting of the American Public Health Association, New York, NY, September 30–October 4, 1990.

References

1. Dirección General de Epidemiología: Situación del SIDA en México hasta el 31 de Diciembre de 1989. Bol Mens SIDA (México) 1989; 4(1):781–787.
2. Valdespino JL, Izazola JA, Rico B: AIDS in Mexico: Trends and projections. PAHO Bull 1989; 23(1-2):20–23.
3. US Department of Justice: Statistical Yearbook of the Immigration and Naturalization Service, Washington, DC, 1987.
4. Mundeshall OS: Elementary Survey Sampling. Belmont, CA: Duxbury Press, 1971.
5. Genetic Systems: Enzyme immunoassay (EIA) for the detection of antibody to human T-lymphotropic virus type III (HTLV III) in human serum or plasma. Seattle, WA: Genetic Systems Corp, 1986.
6. Gallo D, Diggs JL, Shell GR, Dailey PJ, Hoffman MN, Riggs JL: Comparison of detection of antibody to the acquired immunodeficiency syndrome virus by enzyme immunoassay, immunofluorescence, and Western blot methods. J Clin Microbiol 1986; 23:1049–1051.
7. Emmons RW, Riggs JL: Application of immunofluorescence to diagnosis of viral infections. Methods in Virology. New York: Academic Press, 1977.
8. Specter S, Lance GJ: Laboratory diagnosis of human retrovirus infections. Clinical Virology Manual. New York: Elsevier Science Publishing Company, 1986.
9. Fleiss JL: Statistical Methods for Rates and Proportions. New York: John Wiley & Sons, 1981; 168–173.
10. Winkelstein W Jr, *et al:* The San Francisco men's study. III. Reduction in human immunodeficiency virus transmission among homosexual/bisexual men. Am J Public Health 1987; 77:685–689.
11. Joseph JG, *et al:* Magnitude and determinants of behavioral risk reduction: Longitudinal analysis of a cohort at risk for AIDS. Psychol Health 1987; 1:73–96.
12. Martin JL: The impact of AIDS on gay male sexual behavior patterns in New York City. Am J Public Health 1987; 77:578–581.
13. Hahn RA, Onorato IM, Jones TS, Dougherty J: Prevalence of HIV infection among intravenous drug users in the United States. JAMA 1989; 261(18):2677–2684.
14. Winkelstein W, Wiley JA, Padian NS, *et al:* The San Francisco men's health study: Continued decline in HIV seroconversion rates among homosexual/bisexual men. Am J Public Health 1988; 78:1472–1474.
15. Centers for Disease Control: Declining rates of rectal and pharyngeal gonorrhea among males—New York City. MMWR 1984; 33:295–297.
16. Becker MH, Joseph JG: AIDS and behavioral change to reduce risk: A review. Am J Public Health 1988; 78:394–410.

HIV Infection in an Ethiopian Prison

Yohannes Kebede, MD, MPH, Joyce Pickering, MD, Jane C. McDonald, MD, Kay Wotton, MD, MPH, and Debrework Zewde, PhD

ABSTRACT

HIV-1 antibody was detected in 6.0 percent of prisoners in an Ethiopian prison. HIV-1 seropositivity was strongly associated with positive VDRL status (RR = 3.7) and recent admission to prison (RR = 3.5). Seropositive prisoners were more likely to have prostitute contacts. No correlation was found between seropositivity and number of sexual contacts, long-term sex partners, or dental extractions. HIV-1 infection in this area may be of recent origin and the high prevalence may reflect rates in the surrounding community. (*Am J Public Health* 1991;81:625–627)

Introduction

Infection with human immunodeficiency virus (HIV-1) in Africa has increased rapidly. By 1988, 11,753 cases had been reported from 45 countries.[1,2] In Ethiopia, 285 AIDS (acquired immunodeficiency syndrome) cases have been reported to the National AIDS Prevention and Control Unit, by January 1990,[3] the majority (70.4 percent) from Addis Ababa.

This study was conducted in the major prison in Dire Dawa to assess the rate of HIV-1 seropositivity and associated factors. Dire Dawa is on the main communication and trade route linking Addis Ababa to the Red Sea ports of Assab and Djibouti. People in the area have high mobility and contact with neighboring regions and countries. Information obtained will lead to more relevant approaches to health prevention practices in prisons in Ethiopia and to increased awareness of the extent of HIV-1 infection in this region.

Methods

Four hundred fifty consenting prisoners from Dire Dawa District prison, present in November 1988, were enrolled. An oral closed-ended questionnaire was pretested and then administered after standardized translation into Oromingna, Somaligna, and Amharic.

Characteristics including age, sex, education, marital status, and time in prison were determined. Aspects of sexual behavior were obtained during confidential interviews.

All participants had blood samples drawn for HIV-1 and VDRL status. HIV-1 testing was done in the National

---

Address reprint requests to Jane C. McDonald, MD, Division of Infectious Diseases, Montreal Children's Hospital, 2300 Tupper, Montreal, Quebec, H3H 1P3. Dr. Kebede is with the Department of Community Health, Addis Ababa University, Ethiopia; Drs. Pickering, McDonald, and Wotton are affiliated with the Department of Epidemiology and Biostatistics, McGill University, and with the Department of Community Medicine, Addis Ababa University; Dr. Zewde is with the AIDS Laboratory, National Research Institute of Health, Addis Ababa, Ethiopia. Drs. Pickering, McDonald, and Wotton are fellows of the Royal College of Physicians (Canada). This manuscript, received June 19, 1990, was revised and accepted for publication January 23, 1991.

Research Institute of Health (NRIH) in Addis Ababa using the Wellcozyme competitive enzyme linked immunosorbent assay (ELISA) (Wellcozyme anti-HTLV-III, Wellcome Diagnostics, Dartford, UK). All repeatedly reactive specimens were analyzed by Western Blot (Bio-Rad Novapath Immunoblot Assay) and considered positive if bands corresponding to at least one core protein and one envelope protein were visualized.

Syphilis serology was performed using the Venereal Disease Research Lab (VDRL) test.

Associations between HIV-1 seropositivity and sociodemographic factors and risk behaviors were examined using cross-tabulation, chi-square tests, t-test, and odds ratios with their 95 percent confidence limits. Data analysis was carried out using the SPSS statistical program.

Health education was provided to all respondents at the time of the interview. Confidential counseling to all seropositive and seronegative prisoners was offered when results were known. Those with positive VDRL tests were offered Benzathine penicillin G. with all receiving treatment.

Results

During the period November 12–23, 1988, 450 prisoners were enrolled. Of these, 27 (6.0 percent) had positive HIV-1 serology; 141 (31.6 percent) had a positive VDRL titer.

Table 1 compares relevant demographic characteristics in HIV-positive and HIV-negative prisoners. Older prisoners were 2.5 times more likely to be HIV seropositive. There was no significant association between HIV-1 seropositivity and education status, gender, or marital status. A shorter stay (< three months) was significantly associated with HIV-1 seropositivity.

Table 2 compares selected sexual behaviors. Prisoners with prostitute contact before prison were 2.5 times more likely to be HIV-1 seropositive than those prisoners who denied such contact although this did not reach statistical significance. HIV seropositivity was not significantly associated with either the number of long-term sex partners or the frequency of sexual contacts before prison. None of the prisoners reported homosexuality.

Lack of circumcision, and number of injections or dental extractions did not correlate with seropositivity (Table 3).

TABLE 1—Comparison of Selected Demographic Characteristics in HIV-Positive and HIV-Negative Ethiopian Prisoners

Demographics	HIV-positive n = 27 N (%)	HIV-negative n = 423 N (%)	Relative Risk (95% Confidence Interval)
Age (years)			
≤24	8 (4.0)	218 (36.0)	
≥25*	19 (8.5)	205 (91.5)	2.4 (1.07,5.36)
Education			
Illiterate	2 (3.7)	51 (96.3)	
Literacy program	6 (7.5)	74 (92.5)	2.0 (0.42,9.48)
Formal schooling	19 (6.0)	298 (94.0)	1.6 (0.38,6.62)
Duration (years) of Residence in Dire Dawa			
≤5	14 (7.0)	181 (93.0)	1.4 (0.68,2.93)
>5	13 (5.0)	242 (95.0)	
Travel Outside Dire Dawa			
No	4 (8.7)	42 (92.3)	
Yes	23 (5.7)	381 (94.3)	0.6 (0.24,1.81)
Prison Stay (months)			
≤3**	21 (9.3)	205 (90.7)	3.5 (1.43,8.43)
>3	6 (2.7)	218 (97.3)	

*significant variable ($p < 0.05$)
**significant variable ($p < 0.01$)

TABLE 2—Comparison of Selected Sexual Behaviors in HIV-Positive and HIV-Negative Ethiopian Prisoners

Behavior	HIV-positive n (%)	HIV-negative n (%)	Relative Risk (95% Confidence Interval)
Long-term sex partners before prison			
0	9 (6.5)	129 (93.5)	
1	11 (5.8)	177 (94.2)	0.9 (0.38,2.11)
≥2	7 (5.6)	117 (94.4)	0.9 (0.33,2.26)
Sexual contacts per month before prison			
≤1	2 (2.3)	84 (97.7)	
≥2	25 (6.9)	339 (93.1)	2.95 (0.71,12.23)
Prostitute contacts per month before prison			
0	5 (3.1)	156 (96.9)	
≥1	22 (7.6)	267 (92.4)	2.5 (0.95,6.35)

TABLE 3—Number of Injections, Dental Extractions, Immunizations, by HIV Status of Ethiopian Prisoners

	HIV-positive n (%)	HIV-negative n (%)	Relative Risk (95% Confidence Interval)
Injection during past 5 years			
0	9 (5.2)	163 (94.8)	
≥1	18 (6.5)	260 (93.5)	1.2 (0.57,2.69)
Dental extractions in past			
0	22 (6.5)	319 (93.5)	
≥1	5 (4.6)	104 (95.4)	0.7 (0.28,1.83)
Immunizations in past			
No	12 (6.0)	188 (94.0)	
Yes	15 (6.0)	236 (94.0)	1.0 (0.48,2.08)

TABLE 4—VDRL Status and HIV Infection

	HIV-positive n (%)	HIV-negative n (%)	Relative Risk (95% Confidence Interval)
VDRL Status			
Negative	10 (3.2)	299 (96.8)	
Positive*	17 (12.0)	124 (88.0)	3.7 (1.75,7.93)

*significant variable ($p < 0.001$)

The association between positive VDRL status and HIV-1 positivity was significant (Table 4).

To determine whether age was a confounder in the association between HIV positivity and other variables, chi square analyses were performed. The only significant association with age was time in prison such that young prisoners were more likely to have short stays. Because both older age and shorter stay in prison were associated with higher seropositivity rates, the effect of age on the bivariate analysis is to underestimate the shorter prison stay and higher seropositivity association.

Discussion

The seropositive prisoners in this study represent a prevalence of 6.0 percent. Among high-risk groups such as Ethiopian prostitutes, rates from 16 percent to 60 percent have been reported.[4]

Higher rates of HIV infection have been shown in prisoners than the general population. Seropositivity rates of 11 percent, 16.8 percent, and 26 percent have been reported from Switzerland, Italy, and Spain, respectively.[5] The spread of HIV infection in closed institutions is a major public health concern. An estimated 21,000 to 42,000 prisoners were infected with HIV in American State prisons in 1986 with 420 diagnosed cases of AIDS.[6-8] Although no studies have looked at the problem in African prisons, a similar situation is considered likely to exist.

The fact that short duration of stay was associated with seropositivity suggests that HIV was recently introduced into the town. Dire Dawa is on the main truck and trade route between Addis Ababa and the Red Sea, and infection could have been introduced from either direction.

Positive VDRL status was strongly associated with HIV positivity, as has been reported from Nairobi.[11] Men with HIV infection may be more susceptible to syphilis, or syphilis could change the susceptibility of the host by providing a portal of entry for the virus through a genital ulcer. Positive VDRL status may indicate greater exposure to sexually transmitted diseases including HIV[9,10] Genital ulcers, either by history or by examination, were significantly associated with HIV seropositivity in Nairobi.[11] Studies in homosexual populations have confirmed the association of genital ulcers and HIV transmission.[12]

Frequent use of prostitutes has been shown to be associated with HIV seropositivity.[11,13] In our study, a trend was apparent but not significant, possibly because of the small number of seropositive prisoners. Nevertheless, the trend, is suggestive.

A history of injections was not associated with seropositivity in this study. Results from studies in Zaire have been equivocal.[14,15] However, the continuing use of unsterilized needles in many African countries may potentially facilitate HIV transmission.

Immunizations and dental extractions were not associated with HIV seropositivity although the number of positive respondents in this area was small.

In summary, a prevalence rate of 6.0 percent was found in this prison population. The general HIV seroprevalence rate in Ethiopia is approximately 2 percent seropositivity in blood donors.[3] HIV infection appears to have been recently introduced into the area and this high rate is evidence of rapid spread in this group. Seropositivity was significantly associated with positive VDRL status and seropositive men reported more frequent prostitute contacts.

Significant educational efforts are needed to prevent further spread of HIV infection into the surrounding community. ☐

Acknowledgments

This study was supported by a research grant from the International Development Research Center, Canada. Special thanks go to Drs. Francis Aboud, Charles Larson, and Gary Pekeles from the McGill-Ethiopia Community Health Project; the staff and prisoners of the Dire Dawa Prison; the Department of AIDS Control of the Ministry of Health of Ethiopia; and the team of the AIDS Laboratory of the National Research Institute of Health.

References

1. UPDATE: AIDS cases reported to surveillance, forecasting and impact assessment unit. (SFI) Global program on AIDS, June 30, 1988.
2. Mann JM, Chin J: AIDS: A global perspective. N Engl J Med 1988; 319:302–303.
3. National AIDS Prevention and Control Unit (Ethiopia). Acquired Immunodeficiency Syndrome (AIDS): Cases. January 1990.
4. Ayehunie S, Britton S, Yemane-Berman T, *et al:* Prevalence of Anti-HIV antibodies in prostitutes and their clients in Addis Ababa, Ethiopia (Abstract). Scand J Immunol 1987; 26:304.
5. Harding TW: AIDS in prison. Lancet 1987; 2:1260–1264.
6. Berkmals RV: AIDS deaths fuel concern of spread in jails. Am Med News 1986; 29:7.
7. National Institute of Justice and American Correctional Association: Acquired immunodeficiency syndrome in correctional facilities: Issues and options, 1986.
8. Vaid U: National Prison Project Gathers the Facts on AIDS in Prison. ACLU National Prison Project Journal, 1985; 6:1–5.
9. Piot P, Plummer FA, Mhalu FS, *et al:* AIDS: An international perspective. Science 1988; 239:537–579.
10. Kreiss JK, Carael M, Meheus A: Role of sexually transmitted diseases in transmitting human immunodeficiency virus. Genitourin Med 1988; 64:1–2.
11. Simonsen JN, Cameron W, Gakinya N, *et al:* Human immunodeficiency virus infection among men with sexually transmitted diseases: Experience from a center in Africa. N Engl J Med 1988; 319:274–278.
12. Holmberg SD, Gerger AR, Stewart JA, *et al:* Herpesvirus as factors in infection with disease from human immunodeficiency virus (HIV): Evidence that herpes simplex type 2 is a risk factor for HIV infection. Presented at the International Society for Sexually Transmitted Diseases Research, Atlanta, August 2–5, 1987 (abstract).
13. Kreiss JK, Koech D, Plummer FA, *et al:* AIDS virus infection in Nairobi prostitutes: Spread of the epidemic to East Africa. N Engl J Med 1986; 314:414–418.
14. Mann JM, Francis H, Quinn TC, *et al:* HIV seroprevalence among hospital workers in Kinshasha, Zaire. JAMA 1986; 256:3099–3102.
15. N'Galy B, Ryder RW, Bila K, *et al:* Human immunodeficiency virus infection among employees in an African hospital. N Engl J Med 1988; 319:1123–1127.

Comparison of AIDS Knowledge, Attitudes, and Behaviors among Incarcerated Adolescents and a Public School Sample in San Francisco

Ralph J. DiClemente, PhD, Mark M. Lanier, MS, Patricia F. Horan, PhD, and Mark Lodico, PhD

ABSTRACT

Data collected from incarcerated youth (n = 113) and a public school sample (n = 802) demonstrate that both adolescent groups have a high level of AIDS knowledge. Incarcerated youth are less aware of HIV risk-reduction behaviors and report markedly higher rates of HIV risk behaviors. Incarcerated youth are at substantially increased risk of HIV infection relative to their school-based counterparts and should be a primary target of HIV prevention programs. (*Am J Public Health* 1991; 81:628–630)

Introduction

Although studies have identified the prevalence of risk behaviors among incarcerated adolescents,[1-3] as well as their knowledge about acquired immunodeficiency syndrome (AIDS),[4] at present, there are no data available comparing knowledge, attitudes, and risk behaviors between this population and a referent adolescent population. In making such a comparison, it is important to control for geographic site given recent findings indicating geographic variation in adolescents' knowledge and attitudes about AIDS,[5] prevalence of HIV (human immunodeficiency virus)-related risk behaviors,[6,7] and HIV prevalence.[8]

The present study provides a comparison of knowledge, attitudes, and HIV-related risk behaviors between adolescents in a detention facility and those in public schools in San Francisco.

Methods

In February 1988, all students enrolled in family life education classes, a required course, in nine primary high schools in the San Francisco Unified School District were asked to participate in the Centers for Disease Control (CDC) surveillance survey of adolescents' knowledge, attitudes, and risk behaviors.[9] Students completed an anonymous self-report survey during a single class period. It provided demographic information and asked about knowledge of AIDS transmission and prevention, sexual behavior, and intravenous drug use. Adolescents incarcerated at an adolescent detention facility in San Francisco also completed the survey, following the same administration protocol used with the school-based sample.

The age, ethnicity, and gender distributions of the school and incarcerated samples, while representative, demonstrate substantial differences particularly with respect to ethnicity and gender. To control for potential differences attributable to demographic variables when comparing survey responses between the samples, multivariate logistic regression analysis[10] is used to adjust for age, gender, and race.

Results

Response rates for school and incarcerated adolescents were high, 96.7 percent and 91.1 percent, respectively. Demographic characteristics of the samples are described in Table 1.

School and incarcerated adolescents generally demonstrate a high level of AIDS knowledge, with few substantial differences (Table 2). Most of the incarcerated youth correctly identify sharing of intravenous drug needles (86 percent) and sexual intercourse (90 percent) as routes of HIV transmission compared with 89 and 90 percent, respectively, for their school-based peers.

Knowledge of risk-reduction strategies identified informational gaps among both school and incarcerated youth; although incarcerated youth were less knowledgeable. While 85 percent of school youth correctly identified condom use as a means of reducing the risk of HIV transmission, only three-fourths of the incarcerated youth did so. Further, only 62 percent and 56 percent of incarcerated youth recognized that, respectively, sexual abstinence and not having sexual partners who use IV (intravenous) drugs reduce the risk of HIV infection. Among the

---

Address reprint requests to Ralph J. DiClemente, PhD, Center for AIDS Prevention Studies, University of California, 74 New Montgomery Street, Suite 600, San Francisco, CA 94105 and Bayview-Hunter's Point Foundation, Multicultural Inquiry and Research on AIDS (MIRA). Dr. Lodico is also with MIRA; Mr. Lanier is with the School of Criminal Justice, Michigan State University, East Lansing, MI; Dr. Horan is with the Department of Psychology, North Carolina State University, Raleigh, NC. This paper, submitted to the Journal May 8, 1990, was revised and accepted for publication January 23, 1991.

TABLE 1—Demographic Comparison of an Incarcerated and School Sample

	Detention (n = 113)	School (n = 802)	Difference (95% CL)
Gender			
Male	76.4	48.5	27.9 (19.4, 36.4)
Female	23.6	51.5	27.9 (19.4, 36.4)
Age (years)			
≤14	16.1	10.6	5.5 (0, 12.6)
15–16	53.6	74.3	20.7 (11.0, 30.4)
≥17	30.4	15.2	15.2 (6.4, 24.0)
Race			
White	11.0	11.4	0.4 (0, 6.6)
Black	65.1	12.4	52.7 (43.6, 61.8)
Hispanic	10.1	13.7	3.6 (0, 9.6)
Asian	5.5	55.9	50.4 (45.0, 55.8)
Other	8.3	6.6	1.7 (0, 7 .1)

95% CL = 95% Confidence Limits

school-based sample, correct responses were 80 percent and 72 percent, respectively.

Attitudes about AIDS

Both samples strongly endorsed the need for AIDS education (Table 2). Marked differences were identified with respect to perceived risk of HIV infection, as 69.4 percent of incarcerated youth compared with 45.2 percent of the school-based sample perceived themselves susceptible to infection. Awareness of AIDS information resources or where to receive an antibody test was low, although incarcerated youth are more knowledgeable than their school-based peers.

Prevalence of Risk-Taking Behavior

Substantial differences were identified between the samples with respect to prevalence of HIV risk behaviors (Table 3). Almost all incarcerated youth report being sexually experienced compared with approximately 28 percent of the school sample. Most incarcerated youth (73 percent) report two or more sex partners within the past year compared with approximately 8 percent of the school sample. A large proportion of incarcerated youths initiate sex at an early age as approximately 52 and 26 percent of the incarcerated and school samples, respectively, report sexual onset at 12 years of age or younger. Condom use among both samples is inconsistent, although incarcerated youth report being less likely to be consistent condom users. Intravenous drug abuse is prevalent, especially among incarcerated youth, although roughly sim-

TABLE 2—Knowledge about AIDS and Risk-Reduction Strategies

	% Correct		Odds Ratio[a]	95% Confidence Limits
	Detention	School		
Statement				
—You can protect yourself from being infected with the AIDS virus.	80.7	84.5	.56	.31, 1.0
—A person can get AIDS from sharing needles used to inject drugs.	85.6	88.6	.67	.35, 1.3
—You can tell if a person has the AIDS virus by looking at the person.	64.2	70.6	.75	.48, 1.2
—Any person infected with the AIDS virus can infect someone else during sexual intercourse.	89.9	93.5	.56	.26, 1.2
—A pregnant woman who has the AIDS virus can infect her unborn baby.	90.8	89.1	1.3	.63, 2.8
—A person can get AIDS by having sexual intercourse.	89.9	89.8	.88	.42, 1.8
—There is a cure for AIDS.	72.5	76.0	.57	.34, .96
—Only gay men can get AIDS.	88.1	92.2	.59	.29, 1.2
—Blood transfusions are now generally safe.	25.7	31.5	.59	.36, .97
Risk-Reduction Strategies				
People can reduce their chances of becoming infected with the AIDS virus by:				
—not having sexual intercourse	62.4	80.3	.38	.24, .61
—using condoms during sexual intercourse	75.9	84.5	.43	.25, .74
—urinating after sexual intercourse	50.9	53.9	.65	.42, 1.0
—having sexual intercourse with one person not infected with AIDS virus	61.1	69.0	.61	.39, .96
—not having sexual intercourse with a person who uses illegal drugs that can be injected	56.0	71.6	.44	.28, .70
—taking birth control pills	74.3	73.7	.80	.49, 1.3
Attitudes about AIDS				
Should students be taught about AIDS in school?	88.4	89.0	1.05	.50, 2.2
Should a student with AIDS have the right to go to school?	46.4	57.0	.64	.44, .99
Would you be willing to go to class with a student with AIDS?	42.3	48.7	.72	.47, 1.1
Do you think you can get AIDS?	69.4	45.2	2.25	1.4, 3.6
Can you keep from getting AIDS?	81.3	77.3	.91	.51, 1.6
Do you know where to get correct information about AIDS?	60.7	53.2	1.01	.65, 1.6
Do you know where to get tested for the AIDS virus?	54.5	34.7	1.81	1.2, 2.8

[a] Odds ratio adjusted by age, gender, and race.

TABLE 3—Prevalence of Sexual and Drug-related Risk Behaviors

Behavior	% Detention	% School	Odds Ratio[a]	95% Confidence Limits
Proportion sexually active	99.1	28.3	5.5	3.3, 9.4
Number of sex partners				
≥3 Lifetime	84.4	15.1	7.4	4.4, 12.4
≥2 Past year	72.9	8.1	9.4	5.6, 15.7
Age at sexual onset				
≤12 years of age	52.1	25.9	2.5	1.5, 4.4
Proportion reporting:				
Consistent condom use[b]	28.6	37.3	0.6	.35, 1.1
Use intravenous drugs[c]	12.9	3.7	3.3	1.5, 7.4

[a] odds ratio adjusted by age, gender, and race.
[b] among sexually active, defined as "always" using condoms.
[c] proportion reporting lifetime prevalence of intravenous drug use.

ilar proportions of intravenous drug users in each sample report sharing needles.

Discussion

Incarcerated adolescents compared with a school sample demonstrate similar levels of AIDS knowledge, although they are less likely to be knowledgeable of prevention strategies—especially the protective value of condoms. Incarcerated youth were more likely to be sexually active, they initiated sex at an earlier age, report more sexual partners, and a smaller proportion use condoms consistently. This combination of behaviors will greatly increase the risk of HIV infection in this population. Likewise, the higher prevalence of intravenous drug abuse among incarcerated youth will also increase their risk.

The findings indicate an urgent need to implement effective AIDS prevention programs for these high-risk youth. AIDS prevention programs specifically targeted for incarcerated youth will have to overcome particular barriers to behavior change, such as low motivation for self-protection and lack of self-esteem.[11] Without prompt intervention, the potential for rapid spread of HIV infection in this population and transmission to other adolescents and adult populations appears substantial. □

Acknowledgments

This project was funded through the San Francisco Unified School District from a grant provided by the Division of Adolescent and School Health, Centers for Disease Control. Presented, in part, at the National Commission on Correctional Health Care. June 18–20, 1990.

References

1. Alexander-Rodriguez T, Vermund SH: Gonorrhea and syphilis in incarcerated urban adolescents: Prevalence and physical signs. Pediatrics 1987; 80:561–564.
2. Bell TA, Farrow JA, Stamm WE, Critchlow CW, Holmes KK: Sexually transmitted diseases in females in a juvenile detention center. Sex Transm Dis 1985; 12:140–144.
3. Council on Scientific Affairs: Health status of detained and incarcerated youths. JAMA 1990; 263:987–991.
4. Lanier MM, McCarthy BR: AIDS awareness and the impact of AIDS education in juvenile corrections. Criminal Justice 1989; 16:395–411.
5. DiClemente RJ, Zorn J, Temoshok L: Adolescents' knowledge of AIDS near AIDS epicenter. Am J Public Health 1987; 77:876–877.
6. Vermund SH, Hein K, Gayle HG, Cary JM, Thomas PA, Drucker E: Acquired immunodeficiency syndrome among adolescents: Case surveillance profiles in New York City and the rest of the United States. Am J Dis Child 1989; 143:1220–1225.
7. Centers for Disease Control: HIV-related beliefs, knowledge, and behaviors among high school students. MMWR 1988; 37(47):717–721.
8. Burke DS, Brundage JF, Goldenbaum M, Gardner LI, Peterson M, Visintine R, Redfield RR, *et al:* Human immunodeficiency virus infections in teenagers. JAMA 1990; 263:2074–2077.
9. Kann L, Nels[illegible] GD, Jones JT, Kolbe LJ: Establishing a system of complementary school-based surveys to annually assess HIV-related knowledge, beliefs, and behaviors among adolescents. J Sch Health 1989; 59:55–58.
10. Hosmer DW, Lemeshow S: Applied Logistic Regression. New York: John Wiley & Sons, 1989.
11. Rolf J, Nanda J, Thompson L, Ma[illegible]non J, Chandra A, Baldwin J, Delahunt M: Issues in AIDS prevention among juvenile offenders. *In:* Woodruff JO, Doherty D, Garrison-Athey J (eds): Troubled Adolescents and HIV Infection: Issues in Prevention and Treatment. Washington, DC: CASSP Technical Assistance Center, Georgetown University Child Development Center, 1989.

The Donation and Sale of Blood by Intravenous Drug Users

Dale D. Chitwood, PhD, J. Bryan Page, PhD, Mary Comerford, MSPH, James A. Inciardi, PhD, Clyde B. McCoy, PhD, Edward Trapido, ScD, and Shenghan Lai, PhD, MPH

ABSTRACT

In spite of efforts to dissuade intravenous drug users (IVDUs) from donating or selling blood, some continue to do so. As part of a longitudinal study, 915 IVDUs in South Florida were interviewed concerning their history of donation or sale of blood and tested for antibodies to HIV-1 and HTLV-I/II. Approximately 17 percent had either donated or sold blood during 1985 through 1988; most contributors (80.4 percent) sold to commercial blood services. IVDUs who had donated/sold blood were more likely to be male and not in drug treatment than were those who had not contributed blood. IVDUs not in treatment at the time of interview were more likely than IVDUs in treatment to have sold blood. Of those who had donated/sold blood since 1985, 19.6 percent subsequently tested positive for antibodies to HIV-1 and 5.7 percent were positive for antibodies to HTLV-I/II. Increased effort is required to screen prospective donors and sellers, particularly at commercial blood banks. (*Am J Public Health* 1991;81:631–633)

Introduction

Because HIV infectivity precedes antibody formation,[1,2] blood service organizations must rely on self-reported history and physical signs to exclude intravenous drug users (IVDUs) from donor pools. The exclusion of IVDUs is also important because they are at high risk for the human lymphotropic viruses (HTLV-I/II),[3,4] which can be transmitted through infected blood[5,6] but for which blood services do not routinely screen.

In spite of increased donor exclusion efforts, IVDUs continue to donate and sell blood.[7] Nelson and colleagues[8] studied the blood donation histories of a predominately Black cohort of IVDUs from the Baltimore metropolitan area and reported that 27.1 percent had donated or sold blood between 1978 and 1988. Data from other geographic areas have not been reported.

We studied a tri-ethnic cohort of IVDUs in Miami-Dade County, Florida, which has the highest number of cases of AIDS (acquired immunodeficiency syndrome) attributable to parenteral drug use in the Southeastern United States.[9] As part of this investigation, we assessed the seroprevalence of HIV-1 and HTLV-I/II among the study sample, compared IVDUs who donated/sold blood with those who did not, compared donors with sellers, and examined the association of drug treatment with the donation/sale of blood.

Methods

Between June 1987 and August 1988, 915 IVDUs in South Florida were enrolled in a longitudinal study to determine HIV-1 serostatus and associated risk behaviors (22.8 percent were HIV seropositive at baseline). Seven hundred and four participants were recruited from drug treatment facilities in South Florida (eight methadone maintenance clinics, three residential drug-free programs, and one residential detoxification facility). An additional 211 subjects not enrolled in a drug treatment program were recruited directly from the streets. All subjects had used a drug or drugs intravenously at some time since January 1, 1978. All participated on a voluntary basis, gave informed consent, were tested for HIV-1 serostatus, and received pre- and post-test counseling. As part of a structured questionnaire, participants answered questions concerning their most recent donation or sale of blood. All were assured these data would be kept fully confidential and would not be disclosed to anyone including treatment center staff. Participants were paid a fee after completion of venipuncture and interview.

All sera were tested for the presence of antibodies to HIV-1. Residual sera, available on 874 of the study participants, were tested subsequently for antibodies to HTLV-I/II. Because of cross-reactivity, the assay did not differentiate between antibodies to HTLV-I or HTLV-II.

Serum was determined to be positive for antibodies to HIV-1 after two positive ELISAs (enzyme-linked immunosorbent assay) determined with Abbott Enzyme Immunoassay and a confirmatory positive Western Blot following the Centers for Disease Control protocols using viral lysate from Hillcrest Biologicals. A radioimmune assay utilizing an in-house ^{125}I-ionated core protein (p24), purified from human virus, was the antigen for detecting antibody to HTLV-I/II. Serum samples were tested at dilutions of 1:10 and 1:100, which bracket the dilution levels of maximum sensitivity for sera of all titers. Sera were evaluated in terms of percent-bound counts using a high-titered reference se-

---

From the Comprehensive Drug Research Center, Department of Oncology, Biopsychosocial Center for the Study of AIDS, and the Department of Psychiatry, University of Miami School of Medicine, all authors except Inciardi who is with the Division of Criminal Justice, University of Delaware, Newark, DE. Address reprint requests to Dale D. Chitwood, PhD, Research Associate Professor, School of Medicine, University of Miami, 1550 N.W. 10th Avenue, Room 301, Miami, FL 33136. This paper, submitted to the Journal May 9, 1990, was revised and accepted for publication November 20, 1990.

Editor's Note: See also related editorial on page 561 of this issue.

TABLE 1—HIV-1 and HTLV-I/II Seroprevalence and History of Donation/Sale of Blood among IVDUs 1985–1988

	HIV-1*		
	+	–	Prevalence (%)
Never Gave	178	583	23.4
Donated	4	26	13.3
Sold	26	97	21.1
	HTLV-I/II**		
	+	–	Prevalence (%)
Never Gave	74	659	10.1
Donated	1	24	4.0
Sold	7	109	6.0

Four individuals who had sold or donated blood were seropositive for antibodies to both HIV-1 and HTLV-I/II.

*One subject tested indeterminate and was excluded from the analysis.

**Residual blood was not available for HTLV-I/II testing for 41 study participants.

TABLE 2—Multiple Logistic Regression Analysis of Behavior of Selling and/or Donating Blood

	Odds Ratio	95% CI
Age (years)		
<30	1.00	
>30	0.79	(0.55, 1.13)
Gender		
Male	1.00	
Female	0.26	(0.19, 0.36)
Ethnicity		
Black		
No	1.00	
Yes	0.96	(0.65, 1.41)
Hispanic		
No	1.00	
Yes	0.63	(0.38, 1.03)
Treatment Status		
Not in treatment	1.00	
In treatment	0.59	(0.39, 0.90)

rum at 1:100 dilution as the 100 percent standard. Sera were scored as positive if the percent-bound counts were greater than 5 percent at the 1:100 dilution.

Logistic regression analysis was used to evaluate the possible association between demographic characteristics of IVDUs and their behavior regarding the donation or sale of blood since 1985.

Results

Of the 915 IVDUs interviewed, 153 (16.7 percent) reported that they had donated or sold blood since January 1, 1985; four out of five (80.4 percent) of these 153 IVDUs had sold blood. A total of 22.8 percent of the study population tested positive for HIV-1 (Table 1). Of the 153 IVDUs who had contributed blood, 19.6 percent tested positive for antibodies to HIV-1 at the time of interview.

Of the subjects who had donated or sold blood, 5.7 percent of those tested for HTLV-I/II were positive. Serostatus at the time they donated/sold their blood is unknown.

When IVDUs who had donated or sold blood were compared with IVDUs who had not done so in a logistic regression, males and IVDUs who were not in treatment were more likely to have donated/sold blood (Table 2). No differences were observed by ethnic or age group. The demographic characteristics of IVDUs who had donated and those who had sold blood were also compared (data not shown). Donors were differentiated from sellers only by treatment status. Of those in treatment who had sold/donated blood, 68.5 percent had sold; 98.8 percent of the IVDUs from the street had sold rather than donated.

Discussion

Although participation in the study was voluntary, results indicate that the donation and sale of blood by IVDUs is not restricted to the Northeastern United States, and that the sale of plasma or blood accounts for almost all IVDU involvement in the blood supply. The percentage of IVDUs in South Florida (16.7 percent) who donated or sold blood between 1985 and 1988 was virtually identical to the 15.0 percent reported by Nelson and colleagues[8] for a cohort of IVDUs in Baltimore. At least four out of five IVDUs from both Miami (80.4 percent) and Baltimore (88.1 percent) who contributed blood during that time period had sold blood or plasma at a commercial service rather than donated at a voluntary blood center.

Blood services utilize blood screening procedures to ensure that the supply is as safe as possible, but the elimination of high-risk donors remains an unfinished task in the overall plan to protect the blood supply. Since the initiation of blood screening for HIV antibodies and the utilization of the Cohn fractionation process and pasteurization procedures, the risk of a recipient receiving HIV infected blood or blood products has diminished significantly, but has not disappeared.[10–12] Screening for antibodies to HTLV-I/II is not routinely conducted. Several IVDUs who donated or sold blood currently test positive for HIV-1 and/or HTLV-I/II. It must be emphasized that there is no way to ascertain the serostatus of these IVDUs at the time of their last donation or sale. Whether any of the blood contributed by these IVDUs became part of the blood supply is unknown, but it is evident that members of this high-risk group continue to donate/sell blood.

The elimination of the sale of blood by IVDUs is a difficult matter. As long as commercial centers exist, IVDUs will attempt to sell their blood. Two characteristics of commercial blood centers facilitate this selling behavior: commercial blood centers which advertise that they pay cash for blood or plasma attract active drug users who do not have stable sources of income; commercial blood centers often are located in inner-city neighborhoods where poverty and drug use are prevalent.

Commercial blood centers must take a more careful approach in the selection of potential sellers. It is imperative that the screening process at commercial blood services be intensified to identify potential sellers who inject drugs or have a history of IV drug use. Commercial blood centers should employ highly qualified individuals, who have had experience with IVDUs, to screen potential sellers for eligibility. Included in the screening process should be a rigorous check for track marks (scarring caused by repeated injections) on arms and other indications of IV drug use.

Drug treatment programs can play an important role in the reduction in the number of IVDUs who donate or sell blood. Educational intervention with past sellers and donors in treatment could have an immediate positive impact.

Blood services, both voluntary and commercial, have an enormous responsibility to exclude IVDUs from their contributor pool. It is incumbent upon these services to staff the blood centers with highly qualified persons who can identify and exclude high-risk persons who continue to donate or sell blood. ☐

Acknowledgments

Supported in part by grants R01DA04433 from the National Institute on Drug Abuse, and P50MH424-55 from the National Institute of Mental Health.

References

1. Imagawa DT, Lee MH, Wolinsky SM, *et al:* Human immunodeficiency virus type 1 infection in homosexual men who remain seronegative for prolonged periods. N Engl J Med 1989; 320:1458–1462.
2. Ward JW, Holmberg SD, Allen JR, *et al:* Transmission of human immunodeficiency virus (HIV) by blood transfusions screened as negative for HIV antibody. N Engl J Med 1988; 318:473–478.
3. Mildvan D, Des Jarlais D, Sotheran J, *et al:* Prevalence and significance of HTLV-I in a cohort of IV drug users in New York. IV International Conference on AIDS, Stockholm, June 12–16, 1988.
4. Robert-Guroff M, Weiss SH, Giron JA, *et al:* Prevalence of antibodies to HTLV-I, -II, and -III in intravenous drug abusers from an AIDS endemic region. JAMA 1986; 255:3133–3137.
5. Williams AE, Fang CT, Slamon DJ, *et al:* Seroprevalence and epidemiological correlates of HTLV-I infection in US blood donors. Science 1988; 240:643–646.
6. Okochi K: Transmission of HTLV-I (ATLV) through cellular components of blood donors carrying antibodies of the virus. *In:* Petricciani JC, Gust ID, Hoppe PA, Krijen HW (eds): AIDS: The Safety of Blood and Blood Products. New York: John Wiley & Sons, 1987; 231–233.
7. Cleary PD, Singer E, Rogers TF, *et al:* Sociodemographic and behavioral characteristics of HIV antibody-positive blood donors. Am J Public Health 1988; 78:953–957.
8. Nelson KE, Vlahov D, Margolick J, Bernal M, Taylor E: Blood and plasma donations among a cohort of intravenous drug users. JAMA 1990; 263:2194–2197.
9. Centers for Disease Control: HIV/AIDS Surveillance Report. Atlanta: CDC, March 1990.
10. Schorr JB, Berkowitz A, Cumming PD, Katz AJ, Sandler SG: Prevalence of HTLV-III antibody in American blood donors. N Engl J Med 1985; 313:385.
11. Ness PM, Douglas DK, Harper M, Polk BF: Declining prevalence of HIV-seropositive blood donors. N Engl J Med 1990; 321:615.
12. Neumann P, O'Shaughnessy M, Remis R, Tsoukas C, Lepine D, Davis M: Laboratory evidence of active HIV-1 infection in Canadians with hemophilia associated with administration of heat-treated factor VIII. J AIDS 1990; 3:278–281.

Hyperendemic Urban Blastomycosis

ABSTRACT

Within the United States, blastomycosis is primarily endemic in the Mississippi and Ohio River valleys, but reliable figures as to its prevalence and incidence are scarce. A review of pathology department files and medical records from three hospitals in Rockford, Illinois, revealed 32 documented cases of blastomycosis in an eight and one-half year period beginning in March 1981. Twenty-three of these cases were urban since they occurred within the Rockford census tract boundaries—an incidence more than 3.5 times greater than expected in an endemic area. Addresses of patients in 18 of the 23 cases were concentrated at the northeast and southwest ends of the city. Implications are discussed. (*Am J Public Health* 1991;81:633–636)

A. Cesare Manetti, MD, MPH

Introduction

North American Blastomycosis is an elusive, cosmopolitan, systemic infection caused by the dimorphic fungus *Blastomyces dermatitidis.* First described by Gilcrest[1] in 1894, the infection became known as "the Chicago disease" because of the number of cases seen from that area. Later it was referred to as North American blastomycosis as the extent of its wider distribution in the United States and Canada was discovered. Today the disease is correctly and simply referred to as blastomycosis, and its etiologic agent is known to be endemic in Europe, the Middle East, Africa and India. In the Mississippi and Ohio River valleys, where it is primarily endemic, blastomycosis occurs sporadically in humans, dogs and other mammals. Blastomycosis is considered rare, but its true incidence is not known since it is not reportable in most states.

Outbreaks of blastomycosis are seldom observed, and those reported have offered unique opportunities to investigate the source of the infection. The nine outbreaks that have been described to date have mostly been associated with outdoor activities.[2,3] All but one have occurred in non-urban locations, and six were observed in the Great Lakes region. In three of the outbreaks, it was possible to isolate *B. dermatitidis* from rotten wood at exposure sites, mostly in an environment around waterways.[3,4]

One of the most baffling aspects of blastomycosis has been the difficulty encountered in demonstrating where the oganism lives in nature and where humans and animals contract the infection. Any unusual concentration of blastomycosis cases is of interest since it may offer some clues as to the natural habitat of *B. dermatitidis.* This is a report of two hyperendemic foci of blastomycosis in an urban setting.

Methods

Blastomycosis is not a reportable illness in the State of Illinois, nevertheless, a sufficient number of cases came to the attention of the director of the Winnebago County Health Department to suggest the need for an investigation of a possible underlying problem. A search of the files of three Rockford hospital pathology departments and of their respective medical records uncovered 32 cases of blastomycosis. They had all been diagnosed by one or more of three modalities, namely culture, histologic sections and cytologic examina-

---

From the Department of Community Health Sciences, School of Public Health, University of Chicago; and the Department of Pathology, University of Illinois College of Medicine at Rockford. Address reprint requests to A. Cesare Manetti, MD, MPH, P.O. Box 4508, Rockford, IL 61110-4508. This paper, submitted to the Journal May 15, 1990, was revised and accepted for publication November 2, 1990.

TABLE 1—Rockford Hospitals: Blastomycosis Cases [March 1981–August 1989]

Case No.	Specimen (Date/Type)	Census Tract	Age/Race/Sex Occupation	Chief Complaint	Signs, Symptoms and Duration	Initial diagnosis/ Remarks
1	3/81 lung biopsy	2	39, Wh, M factory clerk	Cough	Weight loss, fever	Unresolving pneumonia/ thoracotomy
2	3/81 sputum cytology	28	45, Bl, M factory worker	Cough	Weight loss 5 weeks	Unresolving pneumonia
3	4/81 lung biopsy	13	24, Wh, F secretary	Hemoptysis	Pleural pain	Unresolving pneumonia
4	7/82 skin biopsy	36.01	20, Wh, F	Skin problem		Dermatitis
5	12/82 ear tissue	out of Rockford	25, Wh, M salesman	Ear pain, debility	Weight loss, 3 months	Mastoiditis/ mastoidectomy
6	2/83 bronchial biopsy	out of Rockford	31, Wh, F	Chest pain	Weight loss,	Unresolving pneumonia/ bronchoscopy
7	3/83 bronchial lavage	1.05	31, Wh, M grinder	Cough	Hemoptysis	Unresolving pneumonia
8	3/83 sputum, abscesses	1.05	44, Wh, M teacher	Cough	Weight loss, fever, abscesses lower extremity	Unresolving pneumonia
9	7/83 sputum	out of Rockford	31, Wh*, M factory worker	Cough	Weight loss, fever, chest pain	Unresolving pneumonia
10	12/83 tissue	2	42, Wh, M stock rm clerk	Hemoptysis	Weight loss 4 weeks	Unresolving pneumonia/lobectomy
11	2/85 lung biopsy	out of Rockford	55, Wh, F laborer	Chest pain	7 months	Unresolving pneumonia
12	9/85 lung aspirate	2	33, Wh, M machinist	Cough	Fatigue, chest pain, weight loss	Unresolving pneumonia/suspect blastomycosis
13	4/86 bronchial aspirate	37.03	48, Wh, M mechanic	Cough	Weight loss 2 months	Unresolving pneumonia
14	6/86 lung tissue	1.03	43, Wh, M	Cough	Chills, fever 2 months	Unresolving pneumonia
15	7/86 sputum	out of Rockford	55, Wh, M farmer	Cough	Weight loss, fever	Unresolving pneumonia
16	7/86 lung biopsy	out of Rockford	50, Wh, M factory worker	Cough	Flank pain weeks	Unresolving pneumonia/lung cancer
17	8/86 skin, bone and sputum	out of Rockford	44, Wh, M school coach	Wrist pain	Chest problems months	Synovitis, dermatitis, osteomyelitis, pneumonia
18	8/86 sputum	24	39, Bl, M factory worker	Cough	Chest pain, fever 4 weeks	Unresolving pneumonia/ bronchoscopy
19	4/87 sputum, skin	2	41, Wh, F unemployed	Depression	Chest pain, wt loss, fever 4 months	Psychiatric care, lung infiltrates, skin lesions: dermatitis
20	5/87 sputum	27	47, Bl, M furnace repair	Hemoptysis	Cough, skin lesions	Lung mass, dermatitis
21	10/87 sputum	4.01	89, Wh, M factory worker (retired)	Chest pain		Lung cavity by X-ray
22	11/88 sputum	1.04	38, Wh, F	Hemoptysis	Cough, chills	Unresolving pneumonia
23	12/88 pus, sputum	26	33, Bl, M maintenance worker	Calf abscess	Chills, fever, hemoptysis, weight loss	Abscess pneumonia
24	12/88 sputum	24	26, Bl, M	Cough		Pneumonia
25	1/89 skin biopsy	20	53, Wh, F	Skin lesions	multiple sites	Expired; postmortem miliary blastomycosis
26	2/89 sputum	32	27, Bl, F factory worker	Side pain	Wt loss, fever 5 weeks	Unresolving pneumonia
27	3/89 sputum	out of Rockford	59, Wh, M groundskeeper	Cough	bronchitis, 8 weeks	
28	3/89 sputum	out of Rockford	36, Wh*, F	Cough	Fever, chills	Unresolving pneumonia
29	4/89 joint fluid	34	27, Bl, M construction worker	Elbow pain	Swelling, fever, cough, wt loss 4 months	Osteomyelitis, pneumonia
30	5/89 sputum	32	44, Bl, M unemployed	Cough	Weight loss 6 weeks	Pneumonia
31	7/89 bronchial lavage	5.06	51, Wh, F	Chest pain	Cough, hemoptysis, weight loss	Unresolving pneumonia
32	7/89 trans-bronchial biopsy	22	29, Bl, M	Cough	Chills, fever, weight loss 4 weeks	Unresolving pneumonia

*Hispanic surname

Listing of 32 Rockford blastomycosis cases showing time of diagnosis, census tract and, where applicable, age, race, sex, occupation, chief complaint, signs and symptoms, duration of symptoms at time of initial diagnosis and initial working diagnosis.

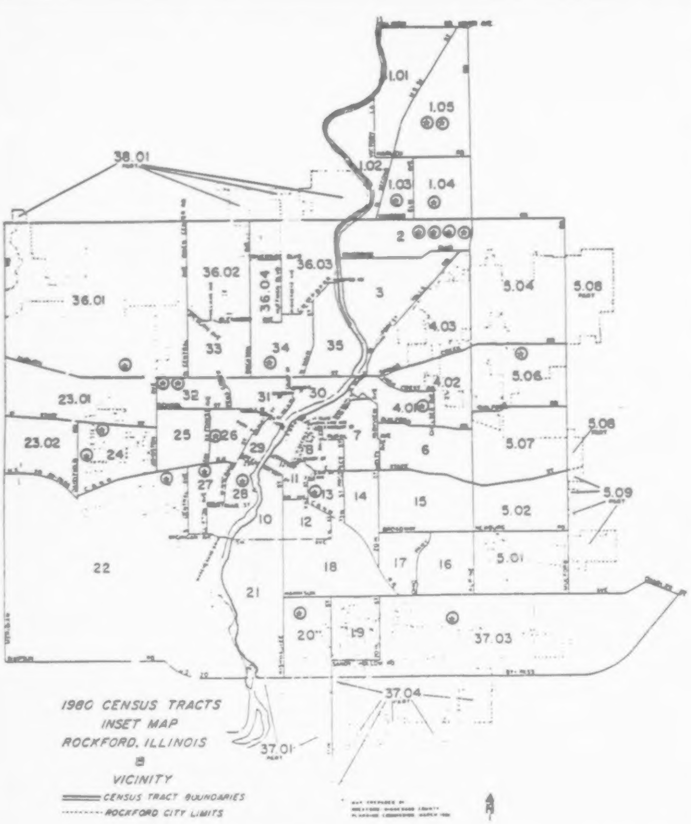


FIGURE 1—1980 Census Tract Inset Map for Rockford and Vicinity in which the Residences of the 23 Urban Rockford Blastomycosis Cases Are Shown Highlighted by a Circled Asterisk.

TABLE 2—Blastomycosis in the City of Rockford (Southwest side) 1980 Population

Census Tract	Total	(Black)	Year	Race	Sex
36.01	2,267	(38)	1982	W	F
34	4,944	(45)	1989	B	M
32	3,965	(1,217)	1989	B	M
			1989	B	F
26	4,452	(2,501)	1988	B	M
24	3,331	(2,402)	1986	B	M
			1988	B	M
22	5,227	(1,960)	1989	B	M
27	3,795	(2,108)	1989	B	M
28	2,153	(1,310)	1981	B	M
Total	30,134	(11,581)		M/F = 8/2	

The 10 cases from one of the two high concentration areas of town are shown according to their specific location, year of occurrence, race and sex. Total and Black population figures per census tract are also given.

tion, during a time span of eight and a half years (from March 1981 to August 1989).

The cases are listed in Table 1 by the month and year when the diagnosis of blastomycosis was first established and by the type of specimen that yielded the diagnosis. The next column gives the census tract in which each Rockford patient lived at the time of initial diagnosis (the patients from other communities are listed as "out-of-Rockford"). Listed in other columns are age, race, sex, occupation, chief complaints, principal signs, symptoms and their duration at the time of initial diagnosis. The initial working diagnosis and other pertinent remarks are given in the last column.

Results

Nine of the 32 patients were referred to Rockford hospitals from outlying areas. Twenty-three patients resided within the census boundaries of Rockford; we will refer to these as the urban areas.

Figure 1 is the 1980 census tract inset map for Rockford and vicinity. In this map, the residences of the 23 urban Rockford cases are highlighted by a circled asterisk.

The distribution of the residences of the 23 Rockford patients was as follows: eight were concentrated in the northeast, 10 were concentrated on the southwest side, and the remaining five were scattered throughout the rest of the city.

The population figures quoted in this study are taken from the 1980 United States Census published in April 1981. According to the Rockford Community Development Department, the population of the city has not appreciably increased or shifted since that time.

Tables 2 and 3 show the 10 cases from the southwest side of town and the eight cases from the northeast side of town according to their census tract, year of occurrence, race and sex, as well as the total population and the black population figures per the 1980 census tract.

Based on the 1980 Rockford census population of 139,712, the annual incidence of blastomycosis in Rockford can be estimated at 1.94/100,000 (23/139,712 × 1/8.5).

Discussion

The following are yearly blastomycosis incidence rates for some states where blastomycosis is endemic[2]:

Arkansas	0.43/100,000
Kentucky	0.44/100,000
Mississippi	0.61/100,000
Wisconsin	0.48/100,000

The estimated incidence rate in Rockford taken as a whole is 3 to 4.5 times the incidence rate for endemic areas, while the estimated incidence in two parts of town is much higher. The annual blastomycosis incidence rates for the south-

TABLE 3—Blastomycosis in the City of Rockford (Northeast side) 1980 Population

Census Tract	Total	(Black)	Year	Race	Sex
1.05	4,242	(81)	1983	W	M
			1983	W	M
1.03	2,654	(5)	1986	W	M
1.04	3,409	(23)	1988	W	F
2	3,807	(8)	1981	W	M
			1983	W	M
			1985	W	M
			1987	W	F
Total	14,112	(117)		M/F = 6/2	

The eight cases from the other high concentration area of town are shown according to their location, year of occurrence, race and sex. Total and Black population figures per census tract are also given.

west side of town is estimated at 3.9/100,000 and that for the northeast area of town is 6.67/100,000—rates which are, respectively, 6 to 9 and 10 to 15 times those reported for endemic blastomycosis.

It should be noted, however, that while the northeast cases all occurred among the predominantly White population, nine out of 10 southwest side cases occurred among the Black minority population, an incidence among Blacks of 9.14/100,000. By contrast, the incidence among Whites inhabiting the same southwest census tract is 0.63/100,000, which is within a reasonable range of the expected endemic incidence.

By the same token, since only five cases of blastomycosis occurred during the same period of time in the population of Rockford living outside of the two high incidence areas, the calculated incidence for these 95,466 individuals is a similar 0.62/100,000.

The present report draws attention to the fact that there can exist foci of a persistent, high concentration of blastomycosis cases in an urban setting. Because of the privileged nature of the hospital records, we were prevented from contacting patients directly to explore further the possible causes of this unusual concentration of cases.

Blastomycosis is a serious, insidious, sometimes indolent disease that frequently goes unrecognized. Eleven of the 50 US states require mandatory reporting of blastomycosis,[5] but many states in a well known endemic area do not, as in the case of Illinois, Indiana, Iowa, Kentucky, Michigan, Missouri, New York and Tennessee.

Given the complexity and serious nature of the condition, we strongly recommend that blastomycosis be included among the reportable infections in Illinois and in neighboring states. ☐

Acknowledgments

This work was prepared in partial requirement for the MPH degree in Community Health Sciences, the University of Illinois, Chicago.

I am indebted to Joseph E. Orthoefer, DVM, for drawing my attention to the problem of Blastomycosis in Rockford. I thank Aquiles Sobrero, MD, Edwin Chen, PhD, and Richard Novak, MD, for their comments on the manuscript. The Winnebago County Health Department and the Medical Record Departments of Rockford Memorial Hospital, Saint Anthony Hospital and Swedish American Hospital were very helpful.

References

1. Gilchrist TC: Protozoan dermatitis. J Cutan Dis 1894; 12:496.
2. Klein BS, Vergeront JM, Davis JP: Epidemiologic aspects of blastomycosis, the enigmatic systemic mycosis. Semin Respir Infect 1986; 1:29–39.
3. Klein BS, Vergeront JM, DiSalvo AF, Kaufman L, Davis JP: Two outbreaks of blastomycosis along rivers in Wisconsin. Am Rev Respir Dis 1987; 136:1333–1338.
4. Klein BS, Vergeront JM, Weeks RJ, *et al:* Isolation of *Blastomyces dermatitidis* in soil associated with a large outbreak of blastomycosis in Wisconsin. N Engl J Med 1986; 314:529–534.
5. Chorba TL, Berkelman RL, Safford SK, Gibbs NP, Hull HF: Mandatory reporting of infectious diseases by clinicians. JAMA 1989; 262:3018–3026.

Use of a Medical Center's Computerized Health Care Database for Notifiable Disease Surveillance

Margaret Watkins, BSN, MPH, Sandra Lapham, MD, MPH, and Wendy Hoy, MB, BScMed

ABSTRACT

The sensitivity of a medical center's inpatient and outpatient database to detect notifiable diseases was examined. Only 53 percent of inpatient and 7 percent of outpatient laboratory-confirmed cases of shigellosis, salmonellosis, giardiasis, and hepatitis were identified by an automated search for matching diagnosis codes. Reasons for lack of sensitivity include nonavailability of laboratory results at the time of diagnosis assignment, use of a standardized encounter form with limited preselected diagnosis codes, and pre-empting of the infectious disease diagnosis by other diagnoses. (*Am J Public Health* 1991; 81:637–639)

Introduction

Most communicable disease surveillance systems in the United States are based on passive reporting by health care providers. Because these have major limitations, such as underreporting and reporting bias,[1,2] investigators continue to search for alternative data sources. Promising among these are computerized hospital discharge databases. Although hospital discharge data have been used for disease surveillance,[3,4] questions remain about their validity and sensitivity. This study examines the sensitivity of one medical center's computerized inpatient and outpatient tracking and billing system when used for notifiable disease surveillance. Also assessed was the medical center's completeness of notifiable disease reporting to the health department.

Methods

Lovelace Medical Center in Albuquerque, New Mexico is a 235-bed hospital and multispecialty group practice with eight "satellite" family practice and urgent care centers. During the study period August 1, 1986–July 31, 1987, Lovelace had approximately 10,000 inpatient admissions and 540,000 outpatient visits. A computerized billing system is used to record International Classification of Diseases, 9th Ed, Clinical Modification (ICD-9-CM)[5] diagnosis codes, as well as demographic, diagnostic and billing information. Inpatient codes are assigned shortly after discharge by Medical Records Department staff, based on providers' documentation in discharge summaries and progress notes. Up to 30 diagnosis codes are captured by the system. Outpatient visit codes (both "scheduled" and "urgent care") are assigned at the time of the visit by the medical provider, using a standardized encounter form with pre-selected diagnostic codes. A maximum of three codes is allowed on the outpatient form.

To determine the sensitivity of the ICD-9-CM coding system and to determine the reporting rates to the health department, laboratory-identified cases of five notifiable diseases were used as the "gold standard." These included shigellosis, salmonellosis, giardiasis, and hepatitis A and B. Inpatient and outpatient databases were searched for matching ICD-9-CM codes, and cases of notifiable diseases reported by Lovelace personnel to the New Mexico Health and Environment Department (NMHED) were ascertained. To assess reasons for discrepancies among these data sources, medical record reviews were performed on cases that were laboratory-identified but not reported to the NMHED (n = 44) and cases that were laboratory-identified but not ICD-identified (n = 207). Forty-two of the former (two records not found) were reviewed. Due to resource limitations, a random sample of 60 (29 percent) of the latter cases were reviewed.

Attempts were made to review all non-laboratory-identified cases that were either identified by ICD-9-CM code search (n = 9) or reported to the NMHED (n = 6).

Results

Sensitivity of ICD-9-CM Diagnosis

Each of the three data sources yielded cases that would have otherwise gone undetected (Figure 1). Only about half the inpatient and only 7 percent of the outpatient laboratory-confirmed cases were identified by the matching ICD-9-CM codes (Table 1). However, the ICD-9-CM search identified nine cases—all among outpatients—which were not laboratory-confirmed. None of these were reported to the NMHED. One was a symptomatic contact of a known case, two were

---

Address reprint requests to Margaret Watkins, BSN, MPH, Research Associate, Clinical Studies Division, Lovelace Medical Foundation, 2441 Ridgecrest SE, Albuquerque, NM 87108. Dr. Lapham is an epidemiologist and Dr. Hoy is director of the Division, both at the Foundation. This paper, submitted to the Journal May 2, 1990, was revised and accepted for publication December 20, 1990.

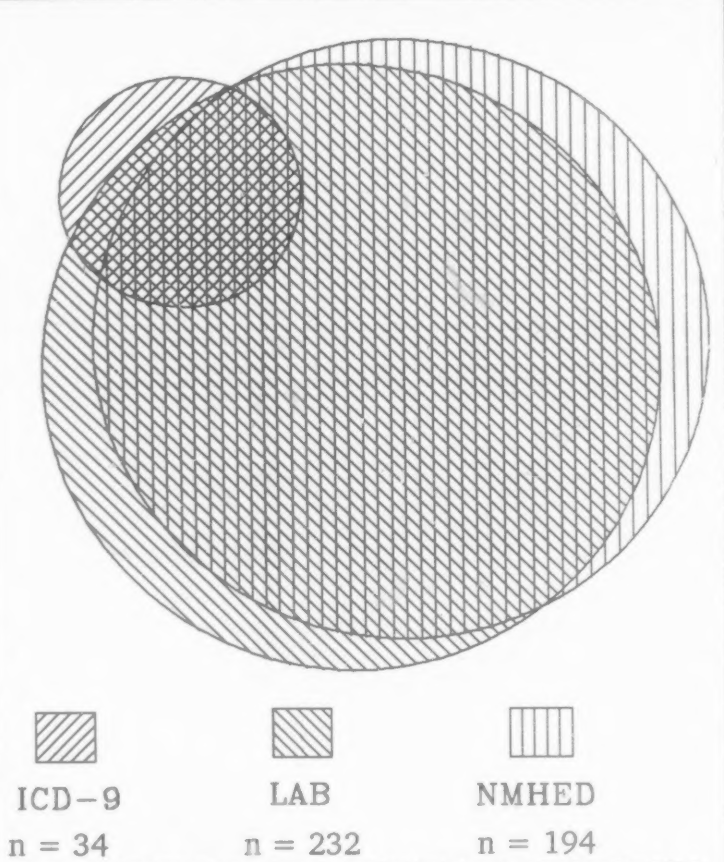


FIGURE 1—Notifiable Disease Cases[a] Identified by Laboratory Confirmation, ICD-9-CM[b] Diagnosis Search Cases Reported to the NMHED,[c] Lovelace Medical Center 8/1/86–7/31/87

a) Salmonellosis, Shigellosis, Giardiasis, Hepatitis A, and Hepatitis B.
b) ICD-9-CM—International Classification of Diseases, 9th ed, Clinical Modification.
c) NMHED—New Mexico Health and Environment Department, Santa Fe, NM.

TABLE 1—Inpatient and Outpatient Laboratory-Confirmed Diagnoses at Lovelace Medical Center: Percent Reported to NMHED[a] and Detected by ICD-9-CM[b] Codes August 1, 1986–July 31, 1987

	Reported to the NMHED[a]		ICD-9-CM[b] Search	
	Inpatient (%)	Outpatient (%)	Inpatient (%)	Outpatient (%)
Salmonella	3/3 (100)	53/54 (98)	2/3 (67)	3/54 (4)
Shigella	1/1 (100)	23/23 (100)	0/1 (0)	1/23 (4)
Giardia	2/2 (100)	25/44 (57)	1/2 (50)	3/44 (7)
Hepatitis A	4/4 (100)	39/55 (71)	3/4 (75)	3/55 (5)
Hepatitis B	9/9 (100)	29/37 (78)	4/9 (44)	6/37 (16)
TOTAL	19/19 (100)	169/213 (79)	10/19 (53)	15/213 (7)
TOTAL inpatient and outpatient	188/232 (81)		25/232 (11)	

[a] NMHED—New Mexico Health and Environmental Department, Santa Fe, NM.
[b] ICD-9-CM—International Classification of Diseases, 9th Ed, Clinical Modification.

chronic or previously diagnosed cases, two were follow-up visits, and one was a case in which the diagnosis—based solely on symptoms—was questionable. (Three records were unattainable.)

Reporting to the Health Department

All inpatient cases and most outpatient laboratory-confirmed cases were reported to the NMHED. Six cases were reported that were not laboratory confirmed; these were symptomatic contacts of known cases or cases in which the diagnosis was based solely on symptoms.

Discussion

Notifiable disease surveillance based solely on ICD-9-CM diagnosis codes was not very sensitive.

In the outpatient setting, miscoding on the initial visit is partly explained by the fact that the laboratory diagnosis has not been established or confirmed. However, medical record review revealed that at the follow-up visit, when positive laboratory results were documented, matching ICD-9-CM codes were still not recorded in approximately half of the cases. Another explanation for the failure to assign appropriate ICD-9-CM codes was the bias introduced by the use of a standardized encounter form with a limited number of preselected diagnosis codes, some of which were nonspecific. The provider seldom uses the option of writing in a diagnosis other than those given on the form. Changes in the encounter form content or usage protocols would be needed before outpatient diagnosis codes would be useful for infectious disease surveillance.

Only half of the inpatient cases were detected by matching ICD-9-CM codes. Review of all the laboratory-identified cases not identified by ICD-9-CM revealed two main reasons for this discrepancy: 1) as with outpatient cases, laboratory results were not always available at the time of discharge, and 2) competing diagnosis; i.e., the infectious disease diagnosis was pre-empted by other diagnoses in complex cases. For example, one patient readmitted with multiple complications after cardiac surgery had 13 discharge diagnoses that did not include his nosocomially-acquired hepatitis B.

Rates of reporting of laboratory-confirmed cases to the NMHED were high. Current policy not only requires that reports of laboratory-confirmed cases are sent to the NMHED, but that salmonella and shigella species isolates are sent to the state scientific laboratory which, in turn, reports to the NMHED. The high rates of reporting of laboratory-confirmed cases documented in this study demonstrate that the system is working efficiently.

Connell, *et al*, have described the opportunities and hazards in the use for research of datasets designed and compiled for other purposes.[6] Our study exemplifies such limitations. Although ICD-9-CM code assignments were not sensitive for detection and surveillance of the notifiable infectious diseases we chose for this study, they were congruent with the clinical picture and may have identified potential cases not detected by laboratory-based surveillance. Conditions whose diagnoses rely predominantly on clinical evidence (e.g., injuries) are likely to be more accurately identified by ICD-9-CM code surveillance. Although further studies on the feasibility of inpatient and outpatient data systems for surveillance are needed, access to a dataset combining laboratory, inpatient, and outpatient information holds potential for disease surveillance. □

Acknowledgments

This work was supported by the Lovelace Medical Foundation and by a Basic Science Research Grant from the National Institutes of Health, NIH-2164-503, and was presented at the 116th Annual Meeting of the American Public Health Association, Boston, MA, November 1988. The authors thank Sarah Valway, DMD, MPH, and Harry Hull, MD, MPH, formerly with the New Mexico Health and Environment Department, for their cooperation and assistance.

References

1. Thacker SB, Choi K, Brachman PS: The surveillance of infectious diseases. JAMA 1983; 249:1181–1185.
2. Alter MJ, Mares A, Hadler SC, Maynard JE: The effect of underreporting on the apparent incidence and epidemiology of acute viral hepatitis. Am J Epidemiol 1987; 125:133–139.
3. Markowitz LE, Hightower AW, Broome CV, Reingold AL: Toxic shock syndrome. JAMA 1987; 258:75–78.
4. Marier R: The reporting of communicable diseases. Am J Epidemiol 1977; 105:587–590.
5. International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM), Volume 1: Diseases: Tabular List. Hyattsville, MD: National Center for Health Statistics, 1980.
6. Connell FA, Diehr P, Hart LG: The use of large data bases in health care studies. Ann Rev Public Health 1987; 8:51–74.

Physical Activity on the Job and Cancer in Missouri

Ross C. Brownson, PhD, Jian C. Chang, PhD, James R. Davis, MS, and Carol A. Smith, MSPH

ABSTRACT

We conducted a series of case-control studies to investigate the risks of 16 cancer types in relation to occupational physical activity. These studies were based on Missouri Cancer Registry data for 17,147 White male cancer patients registered between 1984 and 1989. Colon cancer risk was increased for both the moderate (odds ratio (OR) = 1.1; 95% confidence interval (CI) = 1.0, 1.3) and low (OR = 1.2; 95% CI = 1.0, 1.5) activity levels. Similar elevations were observed for prostate cancer at the moderate (OR = 1.1; 95% CI = 1.0, 1.3) and low (OR = 1.5; 95% CI = 1.2, 1.8) levels of activity, and for cancer of the testis at the low activity level (OR = 2.2; 95% CI = 1.3, 3.7). An opposite trend ($p < 0.01$) was noted for lung cancer, which showed decreased risk at the moderate (OR = 0.9; 95% CI = 0.8, 1.0) and low (OR = 0.8; 95% CI = 0.6, 0.9) activity levels. These associations suggest that further study of the relationship between physical activity and site-specific cancer risk is warranted. (*Am J Public Health* 1991;81:639–642)

Introduction

A growing body of evidence suggests an inverse relation between physical activity and risk of colon cancer. Recent studies,[1–11] from diverse populations, have identified an association between occupational or recreational physical activity and colon cancer risk, although one report found no such relation.[12] Elevations in risk of colon cancer in relation to low physical activity have typically ranged from 20-100 percent.[1–11]

Few studies have evaluated the association between physical activity and other types of cancer. Recent findings suggest that physical activity may be associated with several cancer types including cancer of the stomach,[8] prostate,[9] and breast.[10–13]

To investigate the risks of various cancer types in relation to occupational physical activity, we conducted a series of case-control studies based on data from a statewide cancer registry.

Methods

Subjects were identified through the Missouri Cancer Registry for the time period January 1984 through May 1989. The Registry is maintained by the Missouri Department of Health and has been collecting data on incident cancer cases from public and private hospitals since 1972. Hospital reporting has been mandated by law since 1984. Reporting procedures and validity issues have been discussed in more detail elsewhere.[14]

The current study involved a series of case-control studies that included White male cancer patients who were 20 years of age or older at the time of diagnosis. Men with cancer of ill-defined and unknown primary sites (*International Classification of Diseases for Oncology*[15] (ICD-O) codes 195 and 199) were excluded. Selection was limited to White males due to the small

---

Address reprint requests to Ross C. Brownson, PhD, Director, Division of Chronic Disease Prevention and Health Promotion, Missouri Department of Health, 201 Business Loop 70 West, Columbia, MO 65203. Ms. Smith is also with that Division; Dr. Chang is with the Missouri Cancer Registry; Mr. Davis is with the Bureau of Smoking, Tobacco and Cancer, all with the Missouri Department of Health. This paper, submitted to the Journal June 8, 1990, was revised and accepted for publication October 23, 1990.

TABLE 1—Number of Cancer Patients by Site in Missouri Males, 1984–89

Sites	ICD-O codes		Number of Cases
	Topography	Morphology	
Buccal cavity	140–149		585
Esophagus	150		237
Stomach	151		358
Colon	153		1,838
Rectum	154		812
Pancreas	157		409
Larynx	161		335
Lung	162		4,700
Malignant melanoma		8720/3	143
Prostate	185		2,878
Testis	186		252
Bladder	188		1,080
Kidney/other urinary organs	189		515
Brain/other nervous system	191, 192		332
Non-Hodgkin's lymphoma		*	536
Leukemia		9800/3-9940/3	438
Other cancers			1,699
Total			17,147

*Non-Hodgkin's lymphoma working formulation.[17]

number of other races and the lack of occupational diversity among females.

Data on occupation and tobacco use are routinely abstracted from the hospital medical record at the time of diagnosis for the Missouri Cancer Registry. Hospital registrars record employment information for cancer patients using a standardized protocol. These data are subsequently coded by trained cancer data specialists on the Registry staff using the three-digit 1980 US Census codes.[16] The smoking history of each patient is characterized according to status (i.e., never, former, or current use) and, among current smokers, according to the number of packs smoked per day.

Analysis was limited to 17,147 subjects from the Registry who had codable occupational information. The remaining subjects had no occupational information or noninformative job titles such as "retired."

The classification scheme of Garabrant *et al.*[1] with modifications for Missouri work requirements,[11] was used to categorize occupations according to the level of physical activity. Individual occupations were classified according to whether physical activity is required more than 80 percent of the time (high activity), 20–80 percent of the time (moderate activity), or less than 20 percent of the time (low activity). Subjects were then assigned to one of the three activity levels.

In the analysis for each cancer site, all other cancer registrations formed the control group. For example, in the analysis of the buccal cavity (ICD-O codes 140-149), the case group consisted of 585 patients with this cancer type and the control group consisted of 16,562 "other" cancer patients. The distribution of study subjects by cancer type is shown in Table 1.

Maximum likelihood estimates of the odds ratio (OR)[18] were adjusted for age and smoking, and trend tests were conducted.[19] The highest level of activity was used as the reference category.

In calculating site-specific ORs, colon cancer was excluded from the control group since physical activity has been consistently shown to be a risk factor for colon cancer.[1–11] In addition, because some nonsedentary jobs may be at increased risk of lung cancer due to carcinogenic exposures, ORs for physical activity and lung cancer were adjusted according to *a-priori* job assignments. Occupations were assigned to a high-risk or low-risk category based on a previous study of occupation and lung cancer in Missouri.[20]

Results

Table 2 presents the risks of various cancer types according to the level of occupational physical activity. Elevated colon cancer risk was observed for both the moderate (OR = 1.1; 95% confidence interval (CI) = 1.0, 1.3) and low (OR = 1.2; 95% CI = 1.0, 1.5) levels of activity, with an inverse trend ($p = 0.05$) in risk. Among anatomic subsites of colon cancer, risk was highest for cancer of the cecum, with an OR of 1.3 (95% CI = 0.9, 1.9) at the moderate activity level and an OR of 1.8 (95% CI = 1.0, 3.0) at the low activity level.

Similarly, prostate cancer showed increased risk for moderate (OR = 1.1; 95% CI = 1.0, 1.3) and low (OR = 1.5; 95% = 1.2, 1.8) activity levels. Elevated risk of cancer of the testis was noted for the low activity level (OR = 2.2; 95% CI = 1.3, 3.7). Inverse linear trends were observed for cancers of the prostate ($p < 0.01$) and testis ($p < 0.01$).

An opposite trend ($p < 0.01$) was seen for lung cancer. Decreased risk was shown for moderate (OR = 0.9; 95% CI = 0.8, 1.0) and low (OR = 0.8; 95% CI = 0.6, 0.9) activity levels. Of the major histologic types of lung cancer, the strongest gradient in risk was shown for squamous cell carcinoma. For this cell type, the OR for moderate activity was 1.0 (95% CI = 0.8, 1.1) and the OR for low activity was 0.6 (95% CI = 0.4, 0.8), with a significant gradient in risk ($p < 0.01$).

Discussion

This study corroborates recent studies[1–11] that suggest a relation between low physical activity and colon cancer risk although risk estimates were lower than those reported in some previous studies. Other possible associations were identified between physical activity and cancers of the prostate, testis, and lung.

The physiologic mechanism by which physical activity decreases colon cancer risk is not yet clearly defined but it may relate to increased colon peristalsis[21] and a subsequent decrease in the transit time of the stool and the accompanying exposure of the colon to fecal carcinogens.[4] In addition, experimental animal studies suggest a decreased incidence and growth of induced and transplanted tumors associated with higher levels of physical activity.[22,23]

We found inverse associations between occupational physical activity and cancers of the prostate and testis. Albanes *et al.*[9] recently reported a significant inverse relation between recreational exercise and prostate cancer risk. Previous studies of occupational mortality from Washington State[10] and among San Francisco longshoremen[12] have shown some evidence of a relation between prostate cancer and low job activity. However, other studies[8–12] have shown little evidence of a physical activity-prostate cancer association. The only previous study that investigated physical activity and can-

TABLE 2—Odds Ratios* and 95% Confidence Intervals for Cancer according to Occupational Physical Activity Level in Males, Missouri, 1984–89

Site	Activity Level	Number of Cases	Odds Ratio	95% Confidence Interval	Trend p value
Buccal cavity	moderate	457	1.0	0.8, 1.2	
	low	42	1.1	0.8, 1.7	0.37
Esophagus	moderate	189	1.0	0.7, 1.4	
	low	11	0.7	0.3, 1.4	0.18
Stomach	moderate	281	1.0	0.8, 1.4	
	low	28	1.4	0.9, 2.2	0.13
Colon	moderate	1,476	1.1	1.0, 1.3	
	low	120	1.2	1.0, 1.5	0.05
Rectum	moderate	646	1.1	0.9, 1.3	
	low	55	1.2	0.8, 1.7	0.16
Pancreas	moderate	339	1.3	1.0, 1.8	
	low	22	1.1	0.6, 1.9	0.17
Larynx	moderate	274	1.0	0.7, 1.4	
	low	11	0.5	0.3, 1.0	0.08
Lung	moderate	3,687	0.9	0.8. 1.0	
	low	230	0.8	0.6, 0.9	<0.01
Malignant melanoma	moderate	111	1.0	0.6, 1.6	
	low	11	1.2	0.5, 2.6	0.39
Prostate	moderate	2,301	1.1	1.0, 1.3	
	low	200	1.5	1.2, 1.8	<0.01
Testis	moderate	189	1.1	0.8, 1.7	
	low	31	2.2	1.3, 3.7	<0.01
Bladder	moderate	565	1.1	0.9, 1.3	
	low	66	1.1	0.8, 1.5	0.28
Kidney/other urinary organs	moderate	413	1.2	0.9, 1.5	
	low	36	1.3	0.9, 2.0	0.10
Brain/other nervous system	moderate	256	1.0	0.7, 1.3	
	low	27	1.3	0.8, 2.1	0.27
Non-Hodgkin's lymphoma	moderate	425	1.0	0.8, 1.4	
	low	37	1.2	0.8, 1.8	0.23
Leukemia	moderate	346	1.0	0.8, 1.3	
	low	28	1.1	0.7, 1.7	0.47
Other cancers	moderate	1,384	1.2	1.0, 1.4	
	low	95	1.0	0.8, 1.3	0.24

*Adjusted for age, smoking, and carcinogenic job assignment (lung cancer only); the high activity level was the reference category.

cer of the testis was the study of Harvard and University of Pennsylvania alumni[12] in which no association was observed.

We found a positive association between occupational physical activity and lung cancer risk, although uncontrolled confounding for smoking habits may be partially responsible for this relationship. Earlier studies[8,9,12] have shown a general tendency toward decreased lung cancer risks associated with various measures of higher physical activity levels, although findings have not been entirely consistent.

Severson *et al.*[8] recently reported an increased risk of stomach cancer in relation to several measures of physical activity. These Missouri data showed no evidence of a physical activity-stomach cancer association. We found a slight association between low physical activity and rectal cancer risk, which has been reported inconsistently in previous reports.[1,2,8–10,12]

Our study has several limitations. We used occupational information collected in conjunction with cancer incidence reporting to produce a surrogate measure of physical activity. However, a recent study[14] that compared risk factor information contained in the Registry record with that obtained by interview found reasonable agreement for occupational data. We were also unable to adjust for some potential confounders such as socioeconomic status and dietary history. Although we lacked information on avocational physical activity, it has been reported that the majority of total physical activity in American workers occurs on the job.[24]

Major strengths of our study are the greater accuracy in hospital-reported tumor diagnosis, compared with death certificates,[25] and the ability to analyze data by histologic type or by anatomic subsite.

Our findings for cancers of the prostate, testis, and lung should be considered preliminary and require confirmation in other studies with more detailed measurements of physical activity and potential confounders. □

Acknowledgments

The authors are grateful to the staff of the Missouri Cancer Registry for assistance with data review, coding, and entry. They also thank Dr. David H. Garabrant of the University of Southern California School of Medicine for providing the occupational physical activity classification scheme.

References

1. Garabrant DH, Peters JM, Mack TM, Bernstein L: Job activity and colon cancer risk. Am J Epidemiol 1984; 119:1005–1014.
2. Vena JE, Graham S, Zielezny M: Lifetime occupational exercise and colon cancer. Am J Epidemiol 1985; 122:357–365.
3. Gerhardsson M, Norell SE, Kiviranta H, Pedersen NL, Ahlbom A: Sedentary jobs and colon cancer. Am J Epidemiol 1986; 123:775–780.
4. Wu AH, Paganini-Hill A, Ross RK, Henderson BE: Alcohol, physical activity and

other risk factors for colorectal cancer: a prospective study. Br J Cancer 1987; 55:687–694.

5. Slattery ML, Schumacher MC, Smith KR, West DW, Abd-Elghany N: Physical activity, diet, and risk of colon cancer in Utah. Am J Epidemiol 1988; 128:989–999.
6. Gerhardsson M, Floderus B, Norell SE: Physical activity and colon cancer risk. Int J Epidemiol 1988; 17:743–746.
7. Fredriksson M, Bengtsson N-O, Hardell L, Axelson O: Colon cancer, physical activity, and occupational exposures. A case-control study. Cancer 1989; 63:1838–1842.
8. Severson RK, Nomura AMY, Grove JS, Stemmermann GN: A prospective analysis of physical activity and cancer. Am J Epidemiol 1989; 130:522–529.
9. Albanes D, Blair A, Taylor PR: Physical activity and risk of cancer in the NHANES I population. Am J Public Health 1989; 79:744–750.
10. Vena JE, Graham S, Zielezny M, Brasure J, Swanson MK: Occupational exercise and risk of cancer. Am J Clin Nutr 1987; 45:318–327.
11. Brownson RC, Zahm SH, Chang JC, Blair A: Occupational risk of colon cancer: An analysis by anatomic subsite. Am J Epidemiol 1989; 130:675–687.
12. Paffenbrger RS, Hyde RT, Wing AL: Physical activity and incidence of cancer in diverse populations: A preliminary report. Am J Clin Nutr 1987; 45:312–317.
13. Frisch RE, Wyshak G, Albright NL, *et al:* Lower lifetime occurrence of breast cancer and cancers of the reproductive system among former college athletes. Am J Clin Nutr 1987; 45:328–335.
14. Brownson RC, Davis JR, Chang JC, DiLorenzo TM, Keefe TJ, Bagby JR Jr: A study of the accuracy of cancer risk factor information reported to a central registry compared with that obtained by interview. Am J Epidemiol 1989; 129:616–624.
15. World Health Organization: International Classification of Diseases for Oncology. Geneva: WHO, 1976.
16. US Bureau of the Census: 1980 census of population. Alphabetical index of industries and occupations. Washington, DC: US Govt Printing Office, 1982.
17. The Non-Hodgkin's Lymphoma Pathologic Classification Project: National Cancer Institute sponsored study of classications on non-Hodgkin's lymphomas: Summary and description of a working formulation for clinical usage. Cancer 1982; 49:2112–2135.
18. Gart JJ: Point and interval estimation of the common odds ratio in the combination of 2 × 2 tables with fixed marginals. Biometrika 1970; 57:471–475.
19. Mantel N: Chi-square tests with one degree of freedom, extensions of the Mantel-Haenszel procedure. Am Stat Assoc J 1963; 58:690–700.
20. Zahm SH, Brownson RC, Chang JC, Davis JR: Study of lung cancer histologic types, occupation, and smoking in Missouri. Am J Ind Med 1989; 15:565–578.
21. Holdstock DJ, Misiewicz JJ, Smith T, *et al:* Propulsion (mass movement) in the human colon and its relationship to meals and somatic activity. Gut 1970; 11:91–99.
22. Reddy BS, Sugie S, Lowenfels A: Effect of voluntary exercise on azoymethane-induced colon carcinogenesis in male F344 rats. Cancer Res 1988; 48:7079–7081.
23. Hoffman SA, Paschki KE, DeBias DA, *et al:* The influence of exercise on the growth of transplanted rat tumors. Cancer Res 1962; 4:597–599.
24. LaPorte RE, Cauley JA, Kinsey CM, *et al:* The epidemiology of physical activity in children, college students, middle-aged men, menopausal females, and monkeys. J Chronic Dis 1982; 35:787–795.
25. Percy C, Stanek E III, Gloeckler L: Accuracy of cancer death certificates and its effect on cancer mortality statistics. Am J Public Health 1981; 71:242–250.

Health Care Received by American Children: NCHS Report

The vast majority (83 percent) of American children are covered by health insurance, have a regular source of health care (88 percent, and visited a doctor in the past year for routine health care (64 percent) according to a recent report issued by the National Center for Health Statistics. The report cites new evidence, however, that America's minority and poor children receive less care, including preventive services.

The report—*Health Insurance and Medical Care: Health of Our Nation's Children, United States 1988*—is derived from data from the 1988 National Health Interview Survey on Child Health, conducted by NCHS. Questions on health insurance and sources of medical care were requested for all 17,110 children in a nationally representative sample of children 17 years of age and under.

The survey shows that Black newborns and infants were two to three times more likely to have lacked a routine visit for a check-up or any immunization during the first year of life than were White newborns and infants. Hispanic children were twice as likely to have no health insurance coverage, either public or private, as non-Hispanics.

Most children with a regular source of health care had private care; however, 16 percent of those children (8.7 million) received health care from hospital clinics, emergency rooms, walk-in or emergency centers or other clinics. Clinics were the source of care for 47 percent of Black infants, compared to 16 percent of White infants. For Hispanic infants, almost half received health care in clinics.

The report, *Health Insurance and Medical Care: Health of Our Nation's Children, United States, 1988*—is available from the National Center for Health Statistics, Centers for Disease Control, US Public Health Service, 6525 Belcrest Road, Hyattsville, MD 20782; (301) 436-8500.

Vietnam Era and Vietnam Combat Veterans among the Homeless

Robert Rosenheck, MD, Peggy Gallup, PhD, and Catherine A. Leda, MPH, MSN

ABSTRACT

Of 10,524 homeless veterans assessed in a 43-site VA program, 50 percent served during the Vietnam War era, compared to only 29 percent of all veterans in the general population. This reflects the greater risk of homelessness among men aged 30-44 rather than the impact of Vietnam Era service. The proportion of homeless veterans who served in the Vietnam Theater (44.9 percent), and the proportion exposed to combat fire (40.5 percent) were similar to those of nonhomeless veterans. Homeless combat veterans who are not White were more likely to have psychiatric, alcohol, and medical problems than homeless noncombat Vietnam veterans who are not White. (*Am J Public Health* 1991;81:643–646)

Introduction

Surveys of the homeless have indicated that 43 percent to 50 percent of homeless male veterans served in the military during the Vietnam Era (August, 5, 1964-May 7, 1975),[1-3] compared to only 29 percent of veterans in the general population.[4] During the 1980s, reports of high percentages of Vietnam Era veterans among the homeless appeared virtually simultaneously with a growing body of literature reporting significant post-war readjustment problems among Vietnam combat veterans[5,6] and suggested that veterans who served in Vietnam may be at particularly high risk for homelessness.

Three important shortcomings of these initial studies were: 1) the numbers of Vietnam Era veterans studied were small (53-88 per study); 2) the potentially confounding influence of age was not addressed (Vietnam Era veterans fall into the age cohort that is most at risk for homelessness)[7] and 3) data were not presented on exposure to the presumed combat stressors, whether service in the Vietnam theater of war or exposure to life-threatening combat fire.

In 1987-88, the Department of Veterans Affairs (VA) Homeless Chronically Mentally Ill Veterans Program conducted intake assessments on 10,524 homeless veterans, of whom 5,287 (50 percent) reported service during the Vietnam Era. In this paper, demographic, military service and clinical assessment data on these veterans, and published data on Vietnam Era veterans in the general population, are used to examine: 1) whether Vietnam Era veterans are disproportionately represented among homeless veterans, when age is taken into consideration; 2) whether homeless Vietnam Era veterans are more likely to have served in Vietnam or to have been exposed to combat fire in Vietnam than Vietnam Era veterans in the general population; and 3) whether homeless veterans who served in Vietnam or who were exposed to combat fire there have more frequent medical, psychiatric or substance abuse problems as compared to a reference group of homeless veterans who did not serve in Vietnam.

Methods

Subjects

The Homeless Chronically Mentally Ill (HCMI) Veterans Program was implemented in May 1987, at 43 VA Medical Centers across the country. Sites were selected on the basis of the estimated number of homeless persons in their service area, their capacity to conduct outreach to the homeless and the presence of a coalition of agencies assisting the homeless in their area.

Homeless veterans were contacted through several routes: 52.7 percent were contacted through community outreach efforts in which social workers and nurses visited shelters, soup kitchens and other places frequented by the homeless, seeking out veterans in need of assistance; 26.1 percent came to VA facilities on their own; 13.3 percent were referred by non-VA programs for the homeless; and 7.9 percent came through unspecified routes. Veterans were offered intake assessments if they were judged to be eligible for VA benefits, lacked a conventional place to sleep, and expressed interest in VA health care services or other benefits.

Procedures

A standardized intake form recording data gathered through a structured interview, and clinicians' observations of major psychiatric symptoms, was completed on veterans assessed during the first 10.5 months of program operation (May 15, 1987-March 30, 1988). The interview addressed self-reported age, gender, ethnicity, duration of the current episode of homelessness, dates of admission and discharge from military service, receipt of combat pay, exposure to fire during com-

---

From the Northeast Program Evaluation Center, VA Medical Center, West Haven, CT (all authors); and the Yale University Department of Psychiatry (Rosenheck). Address reprint requests to Robert Rosenheck, MD/182, Northeast Program Evaluation Center, Veterans Administration Medical Center, 950 Campbell Avenue, West Haven, CT 06516. This paper, submitted to the Journal May 2, 1990, was revised and accepted for publication September 19, 1990.

TABLE 1—Percent of Veterans in Five-year Age Cohorts: HCMI Veterans Program (May 15, 1987–March 31, 1988) and All US Veterans, 1987

Age Cohort (Years)	HCMI Veterans Program N = 10,193 +	All U.S. Veterans* N = 28,688,000
	%	%
<25	1.4	1.1
25–29	7.9	3.9
30–34	16.9	5.7
35–39	20.9	8.3
40–44	16.5	12.0
45–49	9.1	8.7
50–54	8.6	9.4
55–59	8.4	12.4
60–64	5.8	14.4
65–69	2.7	12.9
70–74	1.1	6.9
>75	0.5	4.3
Total	100.0	100.0

*VA Annual Report, 1989.
+data missing on 331 veterans.

bat service, and past history of psychiatric and substance abuse hospitalization. Alcohol and drug abuse problems were identified through a combination of patient self-reports (e.g., "Do you have a problem with alcohol dependency now?") and clinician observations. Major psychiatric problems were assessed with a 10-item summed Psychiatric Problem Index (PPI), consisting of three self-report items and seven clinical observations, (Cronbach's alpha = 0.71). On a sub-sample of 1,318 veterans asssessed in this Program, a PPI of two or more was associated with a major DSM-III psychiatric disorder (schizophrenia or other psychosis, bipolar disorder or depressive disorder), as determined by a psychiatrist's examination, with a sensitivity of 80.1 percent and a specificity of 58.0 percent. A score of two or more on the PPI was thus considered evidence of significant psychiatric disorder.

Military service during the Vietnam Era was categorized as follows. All veterans who served between August 7, 1964 and May 5, 1975 are officially classified by VA as *Vietnam Era* veterans. Within this group, *Vietnam Era Only* veterans (those who did not serve in Vietnam, and who were not exposed to warzone stress) were distinguished from *Vietnam Theater* veterans. Within the group of Vietnam Theater veterans, *noncombat exposed* veterans (those who served in Vietnam but were not under fire) were distinguished from *combat exposed* veterans.

Published data on veterans in the general population were used for comparison purposes and included the Census Bureau's 1987 civilian population estimates[4] and the Third Survey of Veterans,[8] a 1987 survey of a national sample of 9,442 veterans.

Comparisons were made between the proportions of homeless veterans in various age and military service categories and estimated proportions of veterans in the general population in those categories. In addition, health status indicators for noncombat-exposed and combat-exposed Vietnam Theater veterans were compared with health status indicators for veterans who did not serve in Vietnam. Measurement precision was expressed as the 95 percent confidence interval of differences in percentages.

Results

Veteran Characteristics

Age Distribution: All Service Eras: The median age among *all* veterans in the HCMI Program was 40.1 years; 54.3 percent were 30-44 years old (Table 1). Homeless veterans in this program were thus similar in age to other homeless Americans who have a median age of 36.1 years.[7] In contrast, the median age among veterans in the general veteran population in 1987 was 54.4 years; only 26 percent were 30-44 years old. Among veterans, as in the general population,[7] the largest percentages of the homeless are in their 30s and early 40s.

Vietnam Era Service: Over half of all veterans in the HCMI Veterans Program served during the Vietnam Era, compared to only 29 percent in the general veteran population. However, within matched five-year age cohorts, the percentages of Vietnam Era veterans in the HCMI Veterans Program were generally similar to those in the general veteran population, although among the homeless, greater percentages of Vietnam Era veterans were found within younger age cohorts (Table 2). Among both homeless veterans and veterans in the general population, over 80 percent of 30-44 year-olds were Vietnam Era veterans (87 percent in the HCMI Veterans Program; 81 percent in the general veteran population).

Characteristics of Vietnam Era Veterans: Vietnam Era veterans were 99 percent male; 54.6 percent White; 36.8 percent Black; 6.9 percent Hispanic; and 1.7 percent other. The racial distribution was similar to that reported in other studies of the homeless.[7] The duration of the current episode of homelessness was less than one month for 25.8 percent, from one month to one year for 43.9 percent and more than one year for 30.3 percent.

Vietnam Theater Service: Among Vietnam Era veterans assessed by the HCMI Veterans Program 44.9 percent reported service in the Vietnam Theater; 40.5 percent reported exposure to combat and 4.4 percent reported no combat exposure. These proportions are quite similar to those of Vietnam Era veterans in the general population, among whom 48.7 percent report service in the Vietnam Theater; 38.4 percent report exposure to combat and 10.3 percent report war zone service but no combat exposure.[8]

The percentage of White homeless Vietnam Era veterans who reported service in the Vietnam Theater was somewhat greater than that of other racial-ethnic groups (46.2 percent vs 42.4 percent). In contrast, in the general population of veterans 48.0 percent of Whites report service in the Vietnam Theater compared to 52.1 percent of other racial-ethnic groups.

Clinical Assessment of Homeless Vietnam Era Veterans: Among homeless Vietnam Theater combat veterans who were not white, current medical, psychiatric and alcohol problems, as well as past histories of psychiatric hospitalization, were more common than among noncombat homeless veterans of similar race-ethnicity (Table 3). While the same trends were apparent among Whites, they were of lesser magnitude.

TABLE 2—Proportion of Veterans Who Served During the Vietnam Era by Five-year Age Cohorts: HCMI Veterans Program (May 15, 1987–March 31, 1988) and All US Veterans, 1987

Age Cohort (Years)	HCMI Veterans Program N = 10,193	All US Veterans N = 28,688,000*
	%	%
<25	2.1	0.0
25–29	2.5	1.1
30–34	66.4	42.0
35–39	95.6	89.9
40–44	91.4	94.3
45–49	40.8	49.6
50–54	6.1	13.7
55–59	2.6	7.9
60–64	0.8	3.6
65–69	0.4	2.4
70–74	0.9	1.8
>75	1.9	1.1
Total (All ages)	51.0	28.9

*VA Annual Report, 1989.
+Data missing on 331 veterans.

TABLE 3—Comparison of Clinical Problems Identified on Screening Assessment between Veterans Who Did Not Serve in Vietnam and Those Exposed to Combat, by Race

	Vietnam Era Only	Vietnam Theater/ Combat Exposed	Difference in Percent (95% CI)
White	N = 1,528	N = 1,189	
	%	%	%
Medical Problems	41.0	44.6	3.5 (−0.2,7.3)
Psychiatric Problem	50.0	52.1	2.1 (−1.7,5.9)
Alcohol Problems	51.2	52.8	1.6 (−2.2,5.4)
Drug Problems	17.1	18.4	1.3 (−1.6,4.2)
Past Psychiatric Hospital	41.5	42.5	1.1 (−2.7,4.8)
Past Alcohol Hospital	50.2	48.9	−1.3 (−5.1,2.5)
Past Drug Hospital	17.7	20.2	2.5 (−0.4,5.5)
Others	N = 1,387	N = 952	
	%	%	%
Medical Problems	37.9	45.8	7.9 (3.7,12.0)
Psychiatric Problem	38.0	48.4	10.5 (6.3,14.6)
Alcohol Problems	44.9	49.1	4.2 (0.0,8.4)
Drug Problems	26.7	26.9	0.2 (−3.5,3.9)
Past Psychiatric Hospital	28.9	35.5	6.6 (2.6,10.5)
Past Alcohol Hospital	38.6	42.4	3.7 (−0.4,7.8)
Past Drug Hospital	26.0	26.3	0.3 (−3.4,4.0)

Discussion

Several methodological limitations of this study require comment. Although the data were collected in a national program, they were not gathered through a systemic sampling procedure and findings cannot be confidently generalized to the national population of homeless Vietnam Era veterans. Both sampling and reporting biases may influence our findings. First, data were collected through a VA clinical program, in which an over-representation of combat-stressed veterans might be expected. Second, because the data were gathered in a VA program, veterans may have tended to overreport their combat experiences, assuming that such reports would improve their access to VA health care services and financial benefits. On the other hand, many Vietnam veterans have been critical of VA services,[9] and some have been reluctant to use them,[10] possibly biasing the representation of Vietnam Theater veterans downward. In our view, Vietnam service is more likely to have been overcounted in this study than undercounted.

The data presented suggest that the large percentage of Vietnam Era veterans among homeless veterans is best explained by the specific vulnerability of men in their 30s and early 40s to homelessness.[7] Neither Vietnam Era service, Vietnam Theater service, nor exposure to combat fire appear to be unexpectedly common among homeless veterans studied.

This conclusion is consistent with findings from the National Vietnam Veterans Readjustment Study (NVVRS),[6] conducted in 1987. The NVVRS did note, however, that veterans who met diagnostic criteria for Posttraumatic Stress Disorder (15.2 percent of all Vietnam Theater veterans) were far more likely to have been homeless than those who did not. While Vietnam service, by itself, does not appear to be a major risk factor for homelessness, those veterans who were most affected by combat may be at greater risk for homelessness than others.

Vietnam veterans in the HCMI Veterans Program who were not White and who were exposed to combat have more frequent psychiatric, medical and alcohol problems than those veterans who are not White who did not serve in Vietnam. These findings are consistent with those of the NVVRS which found substantial differences in health status measures between veterans who were exposed to high levels of combat and those not exposed, and a considerably higher prevalence of Posttraumatic Stress Disorder among those who are not White than among Whites.[6] The absence of significant differences in health status measures among Whites may reflect the limited specificity of our screening instruments, although further research is needed to clarify the meaning of these findings. While not overrepresented among homeless veterans, homeless Vietnam combat veterans, like Vietnam combat veterans examined in other studies, appear to face more frequent medical and psychiatric problems and may require special services. ☐

Acknowledgments

We thank Paul Errera, MD, Boris Astrachan, MD, and Alan Fontana, PhD, for their assistance. The evaluation of the Homeless Chronically Mentally Ill Veterans Program was funded by the US Department of Veterans Affairs. This paper is based on presentations at the annual meeting of the Society for Traumatic Stress Studies, San Francisco, CA, October 25, 1989, and the annual meeting of the American Psychiatric Association, New York, NY, May 15, 1990.

References

1. Farr RK, Koegel P, Burnam A: A Study of Homelessness and Mental Illness in the Skid Row Area of Los Angeles. Los Angeles, CA: Los Angeles County Department of Mental Health, 1986.
2. Gelberg L, Linn LS, Leake: Mental health, alcohol and drug use and criminal history among homeless adults. Am J Psychiatry 1988; 145:191–196.
3. Streuning EL, Pittman J, Rosenblatt A: Characteristics of homeless veterans in the New York City shelter system. *In:* Rosenblatt A (ed): Homelessness. Albany, NY: Rockefeller Institute of Government (in press).
4. Department of Veteran Affairs: Annual Report 1987. Washington DC: Department of Veterans Affairs, 1989.
5. Laufer R, Frey Wouters E, Yager T: Postwar trauma, social and psychological problems of Vietnam veterans in the aftermath of the Vietnam War. *In:* Legacies of Vietnam, Comparative Adjustment of Veterans and Their Peers. Washington DC: US Govt Printing Office, 1981.
6. Kulka R, Schlenger W, Fairbank J, Hough R, Jordan B, Marmar C, Weiss D: National Vietnam Veterans Readjustment Study. Research Triangle Park, NC: Research Triangle Institute, 1988.
7. Rossi P: Down and Out in America, The Causes of Homelessness. Chicago, IL: University of Chicago Press, 1989.
8. Department of Veteran Affairs: 1987 Survey of Veterans. Washington DC: Department of Veterans Affairs, 1989.
9. Myths and Realities: A Study of Attitudes toward Vietnam Era Veterans. Report submitted by the Veterans Administration to the Committee on Veterans Affairs, US Senate, Washington, DC, 1980.
10. Lifton RJ: Home from the War: Vietnam Veterans, Neither Victims nor Executioners. New York: Simon and Schuster, 1973.

Determinants of Late Stage Diagnosis of Breast and Cervical Cancer: The Impact of Age, Race, Social Class, and Hospital Type

Jeanne Mandelblatt, MD, Howard Andrews, PhD, Jon Kerner, PhD, Ann Zauber, PhD, and William Burnett, MD

ABSTRACT

Previous studies of the relationship between cancer stage, age, and race have not controlled for social class and health care setting. Logistic regression analyses, using information from the New York State Tumor Registry and area-level social class indicators, demonstrated that, in New York City, older Black, lower class women in public hospitals were 3.75 and 2.54 times more likely to have late stage breast or cervical cancer, respectively, than were younger White, high social class women in non-public hospitals. (*Am J Public Health* 1991;81:646–649)

Introduction

Age and race have each individually been associated with cancer stage.[1–5] However, previous studies have not controlled for the effects of social class and health care setting. This paper presents findings from logistic regression analyses to quantify the individual and combined effects of age, race, socioeconomic class, and type of health care setting on breast and cervical cancer stage.

Methods

Cancer Data-Tumor Registry

The study sample consisted of all cases of breast and cervical cancer among New York City (NYC) residents that were reported to the New York State Department of Health Tumor Registry between 1980 and 1985. More than 90 percent of incident cases are reported to the registry from NYC.[6] In the study period, there were 22,111 breast cancer, 2,930 invasive cervical cancer, and 6,408 cervical carcinoma in-situ cases with known stages reported. Stage was unknown for 7.5 percent and 2.6 percent of the breast and cervical cases, respectively. Missing stage was associated with age ($p < .0001$) and missing marital status ($p < .01$) for both cancers. Age was classified into five-year categories. Race was reported as either Black or White. There were insufficient data to include Hispanic origin in the analysis. Marital status was dichotomized as "ever" versus "never" married. Hospital type was classed as "public" for the 11 NYC municipal hospitals and "non-public" for the remaining facilities.

Demographic Variables—Census Data

Because individual information on socioeconomic class is not available in registry data, 1980 United States census

---

Address reprint requests to Jeanne Mandelblatt, MD, Assistant Professor, Division of Cancer Control, Department of Epidemiology and Biostatistics, Memorial Sloan-Kettering Cancer Center, Box 60, 1275 York Avenue, New York, NY 10021. Drs. Kerner and Zauber are also with that Department at the Cancer Center; Dr. Andrews is with New York State Psychiatric Institute, New York City; Dr. Burnett is Director, Tumor Registry, New York State Department of Health, Albany. This paper, submitted to the Journal July 12, 1990, was revised and accepted for publication December 5, 1990.

TABLE 1—Sample Characteristics for Cases Used in Each Model

	Model 1	Model 2	Model 3	Model 4
Characteristics	Breast Late vs Early	Breast Distant vs Regional	Cervical Late vs Early	Cervical Invasive vs In-Situ
Number of Cases[a]	19,132	9,991	2,343	7,182
State (% Late)	52.2	NA	45.1	32.6
Age in years, mean (S.D.)	79.5	60.6 (13.92)	52.4 (15.40)	41.7 (15.8)
Race (% Black)	17.6	20.0	41.0	36.8
Hospital (% Public)	6.1	7.7	16.1	14.0
% Ever Married	84.5	84.3	80.4	70.5
% High School[c], mean (S.D.)	62.3 (16.4)	61.0 (16.2)	54.8 (16.2)	57.3 (17.2)
Median Family Income[c], mean (S.D.)	$16,139 (6,427)	$15,755 (6,333)	$12,966 (5,746)	$13,796 (6,190)

a) The number of cases having complete data for all variables.
b) Percent post-menopausal.
c) These variables were added to the NYS Tumor Registry data set using values for the census tract in which the case lived.

TABLE 2—Summary of Logistic Regression Analysis: Prediction of Late Stage Breast Cancer

	Late vs Early		Distant vs Regional	
Variables	Regression Coefficient	Odds Ratio (95% Confidence Interval) (p-value)	Regression Coefficient	Odds Ratio (95% Confidence Interval) (p-value)
Age[a]	.086	1.09 (1.02, 1.17) ($p < .015$)	.086	1.09 (1.08, 1.11) ($p < .001$)
Race (1 = White, 2 = Black)	.215	1.24 (1.14, 1.35) ($p < .001$)	−.083	.92 —[c] ($p < .15$)
Hospital Setting (0 = non-public, 1 = public)	.412	1.51 (1.32, 1.71) ($p < .001$)	.322	1.38 (1.18, 1.62) ($p < .001$)
Marital Status (0 = never, 1 = ever)	−.041	.96 —[c] ($p < .369$)	−.357	.70 (.63, .79) ($p < .001$)
Income[b] ($5,000 increments)	.029	1.03 —[c] ($p < .055$)	−.020	.98 —[c] ($p < .482$)
Education[b] (% high school, 5% increments)	−.051	.95 (.94, .96) ($p < .001$)	−.030	.97 (.95, .99) ($p < .002$)
Intercept	.29		−.96	

a) All age in 5-year intervals, except late vs early breast, which is post vs pre-menopausal.
b) Note: These are continuous variables and the odds ratios represent the increased risk for each incremental increase in the variable, compared to the next lowest level of the variable.
c) Confidence interval includes one.

data on the median family income and the percent of adults with a high school education in the census tract of residence of the case were used as social class measures.

Logistic Regression Models

Two models were developed for each cancer based on groupings with similar treatment patterns. For breast cancer, the first model predicted the outcome of late stage (regional and distant) compared to early stage (localized and in-situ). As a result of recent advances in the treatment of regional disease, a second model was developed comparing distant to regional disease. For cervical cancer, the outcome of the first model was late stage (regional and distant) versus early stage (localized) invasive cancer. In addition, invasive and in-situ disease were compared in a second model as a measure of the success of Pap screening. For all models the independent variables were age, race, marital status, hospital type, income, and education. Logistic regression models were developed using SAS programs. Table 1 presents summary statistics for the cases included in the models.

Results

The logistic regression analysis indicated that, controlling for the other variables, the following factors were associated with late stage breast cancer: postmenopausal age, Black race, low education, and public hospital use (Table 2). Lower income was also weakly associated with late stage ($p < .055$). In the second model, age, public hospital use, marital status, and low education were each associated with an increased risk of distant disease.

Regression coefficients and intercepts from each model were used to calculate the odds of late stage disease for combinations of variables (Table 3). For example, the odds of having distant, compared to regional, breast cancer were 4.77 times higher for a Black woman who was age 70 to 74, unmarried, in the lowest income and education groups, and who used the public hospital system, compared to a White married woman age 40 to 44, in the highest income and education groups, using a non-public hospital.

For invasive cervical cancer, age and public hospital setting were each associated with an increased risk of late stage disease (Table 4). In the second model, age, race, hospital setting, income, and education were each associated with invasive disease.

The composite odds of a 70 to 74 year old, unmarried Black woman in the lowest income and educational group who used the public hospital system having late stage invasive cervical cancer were 2.54 times greater than for a White, married, 40 to 44 year old woman in the highest income and educational levels and using a non-public hospital (Table 5). The odds for

TABLE 3—Risk of Late Stage Breast Cancer for Women of Differing Demographic Profiles

	Late vs Early[a]	Distant vs Regional[b]
	Odds Ratio[c]	Odds Ratio[c]
Profile 1: Base Case[d]		
70–74 years, Black, poor, low education, public hospital, unmarried	3.75	4.77
Profile 2: Young		
40–44 years, Black, poor, low education, public hospital, unmarried	3.43	2.82
Profile 3: White		
70–74 years, White, poor, low education, public hospital, unmarried	3.02	not significant[e]
Profile 4: Non-public hospital		
70–74 years, Black, poor, low education, non-public hospital, unmarried	2.49	3.46
Profile 5: 90% High School		
70–74 years, Black, poor, high education, non-public hospital, unmarried	2.04	3.33

a) The minimum odds for the lowest risk case (a pre-menopausal, White, married woman using a non-public hospital and living in a community where 90% of the adult population completed high school and the median income is $40,000 per year) is .67.

b) The minimum odds for the lowest risk woman (a 40–44 year old, White, married woman using a non-public hospital and living in a community where 90% of the adult population completed high school and the median income is $40,000 per year) is .35.

c) Odds ratios are computed from the division of the maximum odds by the minimum odds. Minimum and maximum odds are calculated using regression coefficients from the logistic models.

d) Low education and education represent living in a community where 30% of adults completed high school and the median income is $10,000 per year.

e) The variable of interest was not significant in the original regression model.

a woman of the same profile having invasive compared to in-situ disease were 39.75 times higher than for her younger, White, non-disadvantaged counterpart.

Discussion

This study demonstrates that elderly, Black, lower socioeconomic status (SES) women who use public hospitals are at extremely high risk of having their breast and cervical cancers diagnosed at late stages. While our findings are consistent with and extend the findings of prior researchers,[1-5] there are two caveats that should be considered in evaluating our conclusions.

First, in our analysis missing stage was associated with age. However, since older women with advanced disease are less likely to be staged than younger women,[7,8] the missing stage information is likely to underestimate the observed effect of age on stage.

Second, since the NY State Tumor Registry does not contain social class indicators, we assigned to each woman the median income and educational level of her community of residence. While we have no way to determine the relationship of the individual and community SES indicators, several studies of cancer incidence and/or mortality have used group-level data to define socioeconomic status[9-15] and have drawn similar conclusions to studies that use individual information.[16-19] Group data can also capture characteristics of the individual's social "context" which may provide information about class gradients that may not be reflected by individual characteristics.[20,21] It is possible that hospital type is serving as such a "contextual" measure in our analysis.

Our findings suggest that cancer control interventions targeted to elderly disadvantaged women, particularly those of color and those who use public hospitals, have the potential to decrease avoidable cancer morbidity and mortality. □

Acknowledgments

The authors would like to thank the New York City Health and Hospitals Corporation for their support of earlier versions of this project.

Portions of this paper were presented at the American Public Health Association 116th Annual Meeting, October 19, 1989, Chicago, Illinois.

References

1. Horm JW, Astire AJ, Young JL, Pollack ES: Cancer Incidence and Mortality in the United States. SEER 1973–81. NIH Pub. No. 85-1837, Bethesda, MD: NIH, 1985.
2. Holmes FF, Hearne E: Cancer stage-to age relationship: Implications for cancer screening in the elderly. J Am Geriatr Soc 1981; 29:55–57.
3. Goodwin JS, Samet JM, Key CR, Humble C, Kutvirt D, Hunt C: Stage at diagnosis of cancer varies with the age of the patient. JAGS 1986; 34:20–26.
4. Freeman HP, Wasfie TJ: Cancer of the breast in poor black women. Cancer 1989; 63:2562–2569.
5. Polednak AP: Breast cancer in Black and White women in New York State. Case distribution and incidence rates by clinical stage at diagnosis. Cancer 1986; 58:807–815.
6. Burnett W: Cancer incidence and mortality by county: New York State. Albany: New York State Dept of Health, 1982.
7. Samet J, Hunt WC, Key C, Humble CG, Goodwin JS: Choice of cancer therapy varies with age of patient. JAMA 1986; 255:3385–3390.
8. Chu J, Diehr P, Feigl P, Glaetke G, Begg C, Clickson A, *et al:* The effect of age on care of women with breast cancer in community hospitals. J Gerontol 1987; 42:185–189.
9. Clementsen J, Nielsen A: The social distribution of cancer in Copenhagen, 1943 to 1947. Br J Cancer 1951; 5:159–171.
10. Cohart EM: Socioeconomic distribution of cancer of the female sex organs in New Haven. Cancer 1955; 8:34–41.
11. Dorn HF, Cutler SJ: Morbidity from cancer in the United States: Parts I and II. Public Health Monogr 1959; 56:1–287.
12. Graham S, Levin M, Lilienfeld AM: The socioeconomic distribution of cancer of various sites in Buffalo, NY, 1948–52. Cancer 1960; 13:180–191.
13. Seidman H: Cancer death rates by site and sex for religious and socioeconomic groups in New York City. Environ Res 1970; 3:234–250.
14. Hoover R, Manson TJ, McKay FW, Fraumeni JF Jr: Geographic patterns of cancer mortality in the United States. *In:* Fraumeni JF Jr (ed): Persons at High Risk of Cancer—An Approach to Cancer Etiology and Control. New York: Academic Press, 1975; 343–360.
15. Dayal HH, Power RN, Chiu C: Race and social economic status in survival from breast cancer. J Chronic Dis 1970; 23:105–116.

16. Logan WP: Social class variations in mortality. Public Health Rep 1954; 69:1217–1223.
17. The Registrar General's decennial supplement, England and Wales, 1961: Occupational mortality tables. London: His Majesty's Stationery Office, 1971.
18. The Registrar General's decennial supplement, England and Wales, 1970–72: Occupational mortality. Series D, No. 1. London: Her Majesty's Stationery Office, 1978.
19. Lilienfeld AM, Levin ML, Kessler II: Cancer in the United States. Cambridge, MA: Harvard University Press, 1972.
20. Krieger N: Race, class, and health: Studies of breast cancer and hypertension; Doctoral dissertation, University of California-Berkeley, 1989.
21. Bassett T, Krieger N: Social class and Black-White differences in breast cancer survival. Am J Public Health 1986; 76:1400–1403.

TABLE 4—Summary of Logistic Regression Analysis: Prediction of Late Stage Cervical Cancer

	Late vs Early		Invasive vs In-Situ	
Variables	Regression Coefficient	Odds Ratio (95% Confidence Interval) (p-value)	Regression Coefficient	Odds Ratio (95% Confidence Interval) (p-value)
Age[a] (5 year increments)	.113	1.12 (1.09, 1.15) (p < .001)	.358	1.43 (1.40, 1.46) (p < .001)
Race (1 = White)	.113	1.12 —[b] (p < .118)	.148	1.16 (1.02, 1.31) (p < .020)
Hospital Setting (0 = non-public, 1 = public)	.247	1.28 (1.02, 1.[illegible]0) (p < .035)	.223	1.25 (1.07, 1.48) (p < .006)
Marital Status (0 = never, 1 = ever)	−.128	.88 —[b] (p < .256)	.104	1.11 —[b] (p < .144)
Income[a] ($5,000 increments)	.000	1.00 —[b] (p < .927)	−.128	.88 (.81, .95) (p < .022)
Education[a] (% high school, 5% increments)	.000	1.00 —[b] (p < .945)	−.030	.97 (.94, .97) (p < .001)
Intercept	−1.66		−.382	

a) Note: These are continuous variables and the odds ratios represent the increased risk for each level compared to the next lowest level.
b) Confidence interval includes one.

TABLE 5—Risk of Late Stage Cervical Cancer for Women of Differing Demographic Profiles

	Late vs Early[a]	Invasive vs In-Situ[b]
	Odds Ratio[e]	Odds Ratio[e]
Profile 1: Base Case[c] 70–74 years, Black, poor, low education, public hospital, unmarried	2.54	39.75
Profile 2: Young 40–44 years, Black, poor, low education, public hospital, unmarried	1.28	4.6
Profile 3: White 70–74 years, White, poor, low education, public hospital, unmarried	not significant[d]	34.33
Profile 4: Non-public hospital 70–74 years, Black, poor, low education, non-public hospital, unmarried	1.99	31.69
Profile 5: 90% High School 70–74 years, Black, poor, high education, non-public hospital, unmarried	not significant	27.40
Profile 6: $40,000 income 70–74 years, Black, high income, low education, non-public hospital, unmarried	not significant	18.16

a) The minimum odds for the lowest risk case (a pre-menopausal, White, married woman using a non-public hospital and living in a community where 90% of the adult population completed high school and the median income is $40,000 per year) is .67.
b) The minimum odds for the lowest risk women (a 40–44 year old, White, married woman using a non-public hospital and living in a community where 90% of the adult population completed high school and the median income is $40,000 per year) is .35.
c) Low education and education represent living in a community where 30% of adults completed high school and the median income is $10,000 per year.
d) The variable of interest was not significant in the original regression model.
e) Odds ratios are computed from the division of the maximum odds by the minimum odds. Minimum and maximum odds are calculated using regression coefficients from the logistic models.

Public Health and the Law

The Effect of Mandatory Premarital HIV Testing on Marriage: The Case of Illinois

Background

In the past 10 years, AIDS (acquired immunodeficiency syndrome) has become a dominant public health concern. During the late 1980s, state governments took steps to counter the health threat of AIDS. One of the most controversial was the requirement of the State of Illinois that applicants for marriage licenses be tested for human immunodeficiency virus (HIV) antibodies. Among the states, Illinois and Louisiana adopted mandatory premarital testing; only in Illinois was a law implemented for a significant length of time.[1]

Effective January 1, 1988, the Illinois marriage law was amended to require a physician's statement on HIV testing be submitted to the county clerk before a marriage license could be issued. The statement affirmed that both parties to the marriage were tested for HIV antibodies and advised of the test results. The HIV testing requirement was one of a number of AIDS-related measures adopted by the state legislature. In this case, the feeling that people getting married ought to know about HIV infection and the absence of a direct cost to the state was persuasive to lawmakers.[2] The HIV testing requirement was repealed in September 1989.

At the time it was introduced, premarital testing was controversial. Mandatory testing was opposed by the Centers for Disease Control (CDC) and various medical groups.[3,4] Simulation indicated that mandatory premarital testing would be very expensive[5] and probably no more cost-effective for case identification than testing for syphilis.[6]

Upon implementation, the HIV testing requirement was said to depress the frequency of marriages in Illinois. Media reports indicated that couples were crossing state lines to avoid the test,[7] although some observers thought the drop in marriages might be permanent:

> It is ironic that the Illinois screening program has discouraged many people from entering into a legal commitment that fosters a mutual monogamous relationship.[8,9]

This report investigates the hypotheses: (a) that mandatory premarital HIV testing decreased marriages in Illinois; and (b) that the requirement increased crossover marriages in states bordering Illinois. Additional analyses investigated the claim that the frequency of marriages in Illinois has rebounded now that premarital testing is no longer mandatory.[10]

Methods

Illinois Premarital Testing Law

For roughly 20 months, from January 1, 1988 through September 10, 1989, Illinois marriage law required a physician's statement that both parties to the proposed marriage were tested for HIV antibodies and advised of the test results be submitted to the county clerk before a marriage license could be issued.[11] The tests must have been administered within 30 days of the license application. The license was good for 60 days. Positive results on the initial enzyme-linked immunosorbent assay (ELISA) test were to be followed by the Western Blot Assay. A person was considered seropositive for HIV virus if he or she was positive on both tests. Seropositive results were to be reported to the Illinois Department of Public Health.[12]

Table 1 presents marriage license requirements for the seven states included in this study at the time the Illinois premarital testing law became effective.[13] Illinois, like two of the bordering and two of the comparison states, required a blood test for syphilis. In Illinois the marriage license became effective the next day; all the other states had longer waiting periods, although in two bordering and two comparison states, these periods could be waived.

TABLE 1—Marriage License Requirements in Seven States, 1987

State	Blood Test	Waiting Period
Illinois	Yes	Marry next day
Indiana	Yes	72 hours
Wisconsin	Yes	5 days, waivable
Missouri	No	3 days, waivable
Ohio	Yes	5 days
Minnesota	No	5 days, waivable
Kansas	Yes	3 days, waivable

Marriage Time Series

Monthly marriage times series were investigated for seven states for the period from January 1980 through August 1989[14]: (a) Illinois; (b) Indiana, Wisconsin, and Missouri, the three states bordering Illinois largest MSAs (metropolitan statistical areas) that would be expected to show crossover increases; and (c) Ohio, Minnesota, and Kansas, three comparison states

---

Address reprint requests to Jack McKillip, PhD, Professor of Psychology, Department of Psychology, Southern Illinois University at Carbondale, Carbondale, IL 62901. This paper was accepted for publication on August 30, 1990 by George J. Annas, JD, MPH, editor of the Public Health & the Law column, and Professor of Health Law, Boston University, School of Public Health, 80 East Concord Street, Boston, MA 02118.

that do not border Illinois and were not expected to show crossover effects. The beginning of the study period was chosen to allow a long enough pre-HIV-testing series to estimate cyclical trends. August 1989 was selected as the final observation because the law was repealed in mid-September 1989.

Monthly marriage frequencies are collected by state health officials and reported to the National Center for Health Statistics (NCHS) which publishes them on a monthly, "provisional" basis. These time series are labeled "provisional" because non-regular reporting schedules across the states require occasional adjustments to published data. During the first half of the study period, these adjustments were noted in the reports. This practice has not been maintained. Marriages are reported by state of occurrence, not by the state in which couples reside.

Primary NCHS interest is in aggregate data, not in maintaining monthly series on a state-by-state basis. Experience indicates that adjustments more likely reflect a random pattern than consistent practice in any of the states.[15] This type of variation in the time series represents a threat of Type II (unreliability) rather than Type I error (history or instrumentation) for the present study. Because of the statistical analysis procedures, monthly time series were used rather than the more stable but less diagnostic yearly time series.

Statistical Analyses

Box-Jenkins ARIMA interrupted times-series analysis[16] was used because of its ability to estimate cyclical trends and to provide unbiased statistical tests of the impact of the premarital testing law. Using this technique, trend and serial dependency parameters first were estimated for the 96 pre-HIV-testing months, January 1980 to December 1987, for each of the seven states. Next an interruption model is tested for the full time series (including the 20 months from January 1988 to August 1989 when HIV testing was required in Illinois). In addition to trend and serial dependency components, the interruption model includes a binary time series (e.g., 000011111) with 0 values for months during the pre-HIV-testing period and with 1 values for testing months. This Testing Law parameter estimates the size and form (if any) of change in the marriage time series at the point when mandatory premarital HIV testing was introduced in Illinois.

Both permanent/temporary, and abrupt/gradual interruption models were tested for all seven states. Only the permanent, abrupt model results are reported (where appropriate) because only this model yielded statistically significant and appropriate testing law parameters. A permanent, abrupt interruption model shifts the level of the time series only at the point of intervention. This shift is maintained for the duration of the intervention.

Three measures of goodness of fit are available for testing models in Box-Jenkins ARIMA times-series analysis,[16] two of which have counterparts in normal multiple regression analysis. Individual parameters are tested for statistical significance by t-tests and overall R^2 is computed for the full models. This R^2 value is generally much larger for times series analysis than is typical for normal multiple regression analysis. A chi-square test is also made of the residual autocorrelation function. This test estimates whether reliable variation remains among the time series observations. Statistical analyses used the 1984 version of the BMDP 2T Box-Jenkins Times Series program.[17]

Results

Decrease of Marriages in Illinois

Figure 1 presents the raw marriage time series for Illinois. Note the recurrent seasonal cycle, marriages up in Summer and down in Winter. The first hypothesis asked whether the time series moved down as a result of premarital HIV testing. Inspecting Figure 1, this hypothesis expects that marriage frequency is lower starting January 1988 than before this date.

The top row of Table 2 presents the results of statistical analyses relevant to the hypothesized drop in marriages in Illinois. The statistically significant seasonal trend component indicates that, over the 10 years studied, marriages in Illinois decreased by 241 a month on a yearly basis (e.g., January 1980 to January 1981). The model also has statistically significant moving average and autoregressive components that are not presented. The statistically significant testing law parameter reveals an abrupt decrease of 1,043 marriages in Illinois in January 1988 when premarital HIV testing became mandatory.

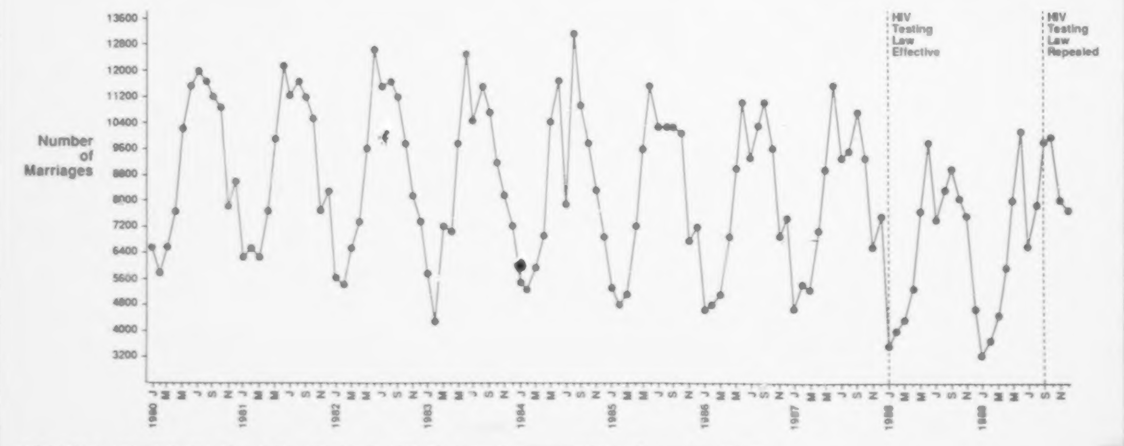


FIGURE 1—Monthly Marriage Time Series for Illinois from January 1980 to December 1989

TABLE 2—Interrupted Time Series Analysis for Marriages in Illinois, as well as Bordering and Comparison States

State	ARIMA Trend12	Testing Law[a]	Model LBQ[b]	Model R^2
Illinois	−241	−1043	26	.941
Indiana	−103	457	19	.857
Wisconsin	−43	324	26	.948
Missouri	−88	317	25	.601
Ohio	−49		22	.945
Minnesota	−52		15	.908
Kansas			19	.826

NOTE: Only statistically significant parameter values are presented. All seven states showed significant serial dependency parameters.

[a] Abrupt, permanent change in monthly marriage time series beginning January 1988.

[b] Chi Square test of ACF residuals at 25 lags. None of the reported values are statistically significant, indicating random residuals.

This monthly drop persisted through August 1989. The model indicates that premarital testing reduced the number of marriages in Illinois by 20,860, about 14 percent over the study period.

Crossover Marriages in Border and Comparison States

Table 2 also presents the results of statistical analyses of marriage time series for the three bordering and three comparison states. An hypothesized crossover effect would be revealed by increases in marriages in the border states and no change in marriages in the comparison states.

Inspection of Table 2 reveals that five of the border and comparison states experienced statistically significant year to year (trend) decline in the frequency of marriage. Kansas was an exception. In that state, monthly marriage frequency cycled around a stable average (level) over the entire study period. All six states showed significant moving average components.

Tests of the crossover hypothesis are provided by the Testing Law parameter. In each of the three border states, analysis revealed an abrupt, permanent, and statistically significant increase in marriages in January 1988. Indiana showed a 10 percent increase, Wisconsin a 9 percent increase, and Missouri an 8 percent increase. Together these increases came to an increase of 1,098 marriages per month, starting in January 1988 and lasting through August 1989. The combined increase in these bordering states almost exactly equals the decrease experienced in Illinois.

Of equal importance for testing the hypothesized crossover effect is the finding that none of the comparison states experienced significant changes in marriages in January 1988. The Testing Law parameter was not significant for any of the comparison states although model prediction (R^2) was as good or better for the comparison states as for the border states.

Rebound of Illinois Marriages

The question of a rebound in the Illinois marriage time series with the repeal of mandatory premarital testing cannot be addressed directly with Box-Jenkins times series analysis without a lengthy, post-law time series. An indirect analysis was made by using the intervention model developed in this study to make predictions for the post-law Illinois experience for the last quarter (October–December) of 1989. This model does not include a rebound parameter. Model predictions were compared with the actual number of marriages reported for Illinois during the final quarter of 1989. A rebound would be observed if the actual number of marriages were greater than those predicted by the interruption model. As shown in Table 3, the no rebound model predicts significantly fewer marriages than actually occurred for each of the three months. The interruption model underpredicted the number of marriages in Illinois by an average of 1,100 per month during the period since the HIV testing requirement was repealed. The rebound in marriages in Illinois was approximately equal to the original drop caused by the testing law.

Discussion

The Illinois premarital testing law produced an abrupt decrease in the number of marriages in Illinois. Analyses revealed a drop in the Illinois marriage time series in January 1988 when the law became effective; the drop occurred immediately and lasted throughout the study period. While severe, about 14 percent, the drop was not as great as the 22.5 percent originally reported.[12] There was no statistical evidence that the drop was temporary or that it diminished or increased over time.

Although there was a significant drop in marriages in Illinois, there was not the predicted absolute drop in marriages. Rather than (a) take the HIV blood test and get married in Illinois, or (b) not get married at all, couples crossed the state line and were married in states bordering Illinois. Analyses reveal a coincident, and approximately equal, abrupt increase in the marriage times series for the states most likely to get marriage crossovers from Illinois—Indiana, Wisconsin and Missouri. These increases did not represent a midwestern trend since January 1988 increases were not found for Ohio, Minnesota, or Kansas.

Analysis suggests that the changes in Illinois marriage patterns produced by the HIV testing requirement were not permanent. With the repeal of the HIV testing law, marriages appear to have rebounded in Illinois.

Cost of HIV Testing

Considerable attention focused on the cost of the HIV test itself as a reason for couples' decisions to cross state lines to marry.[9] The media reported charges of up to $400 a couple for HIV tests.[7,18] However, the financial cost of testing may present too easy an explanation. Turnock and Kelly[12] cite a survey of Illinois hospitals with a mean cost of $30 for each HIV test. For half these hospitals, the charge covered both the ELISA and the Western Blot tests. Even a $30 charge may be high since most couples (99.4 percent) would only require the ELISA test.[5] Mercola[19] reports that the cost of the ELISA test to physicians in Illinois was only $10.

Aside from the financial burden, HIV testing did not increase significantly the difficulty of obtaining a marriage license in Illinois. During this period Illinois required a blood test for syphilis, but so did Wisconsin and Indiana. In Illinois, a marriage license was valid the next day, while a three-to-five-day waiting period was required in the three bordering states (see Table 1).

In order for the cost of the HIV test to have produced the marriage patterns revealed in this report, couples must certainly have incurred logistical and financial expenses greater than the cost of two HIV tests. For example, when an Illinois marriage license cost $15 and required HIV tests, more than 6,000 couples chose instead to be married in Wisconsin. There, a marriage license cost $50 with an additional $10 fee for waiving the five-day waiting period, if the Wisconsin county clerk was willing to grant it.[20]

Fear of AIDS

An alternate explanation for the changes in marriage patterns is fear of an AIDS diagnosis.[12,21] Couples may have

TABLE 3—Study Model Predictions for Post-Premarital Testing Marriage Frequencies in Illinois

1989 Month	No-Rebound Model Predictions	Actual Marriages	Residual (under Prediction)
October	8974	9909	(935)
November	6405	7927	(1522)
December	6771	7615	(844)
Mean	7383	8484	(1100)

NOTE: Standard error for predictions is 664. Residuals indicate that actual marriages were higher than expected from the No-Rebound model.

wanted to avoid learning of their (positive) HIV status. Some evidence supporting this explanation is provided for men by the rise in their seroprevalence rates over the testing period. Turnock and Kelly[12] reported male, marriage license applicants' seroprevalence rates of .85 per 10,000 during the first six months of testing. The Illinois Department of Public Health[22] has released figures indicating male seroprevalence rates of 3.23 per 10,000 over the final 14.3 months of mandatory premarital testing. By contrast, the seroprevalence rate for women remained at about 1.4 per 10,000 during the entire period. Male seroprevalence rates show a four-fold increase from the first six months to the final 14 months of testing suggesting that, at least at the beginning of the testing period, men were avoiding marriage in Illinois because of fear of a positive test result.

Although there is evidence that some men got married outside of Illinois to avoid HIV testing, this explanation does not fit with the large change in marriage patterns any better than does explanation in terms of the cost of the HIV test. One expects that only relatively few men had realistic fears of HIV infection. An explanation that is consistent with the fear of an HIV test result is a generalized fear of AIDS. Perhaps AIDS had become such a specter that the issue itself is to be avoided.[23] Couples may have chosen a way around this unhappy topic by being married in a state that allowed them to without testing. ☐

Jack McKillip, PhD

Acknowledgments

An earlier version was read at the meetings of the Midwestern Psychological Association, Chicago, Illinois, May 5, 1990. Thanks to Tamara Kinkner for help in data preparation and background research and to Lew Hendrix for comments on a previous draft.

References

1. Gostin LO: Public health strategies for confronting AIDS. JAMA 1989; 261:1621–1629.
2. Franklin T: Senate approves AIDS testing for couples planning to marry. Chicago Tribune June 28, 1987; 2, 3:4.
3. Davidson J: AIDS council vows to fight testing plan. Chicago Tribune April 10, 1987; 2, 4:1.
4. Griffin JL: Premarital AIDS test called a waste. Chicago Tribune September 27, 1987; 1, 1:1.
5. Cleary D, Barry MJ, Mayer KH, Brandt AM, Gostin LO, Fineberg HV: Compulsory premarital screening for the human immunodeficiency virus. JAMA 1987; 258:1757–1761.
6. Haskell RJ: A cost-benefit analysis of California's mandatory premarital screening program for syphilis. Western Med 1984; 141:538–541.
7. Enstad R, Jamison P: Marrying kind cross states lines. Chicago Tribune February 5, 1988; 2, 1:1.
8. Belongia EA, Vergeront JM, Davis JP: Premarital HIV screening—In reply. (letter) JAMA 1989; 261:2198.
9. Goodman RD: In sickness or in health: The right to marry and the case of HIV antibody testing. DePaul Law Rev 1989; 38:87–126.
10. Papajohn G, Mount C: Illinois wedding licenses soar after repeal of AIDS test law. Chicago Tribune October 11, 1989; 2, 2:4.
11. Ill. Rev. Stat: Ch. 40, Para 201–219. St. Paul: West Publishing Co, 1988.
12. Turnock BJ, Kelly CJ: Mandatory premarital testing for human immunodeficiency virus: The Illinois experience. JAMA 1989; 261:3415–3418.
13. Skoloff GN: Marriage laws. The world almanac and book of facts. New York: Pharos Books, 1988; 797.
14. National Center for Health Statistics: Births, marriages, divorces and deaths for. . . . Monthly Vital Statistics Report, vol. 29–38. Hyattsville, MD: NCHS, Public Health Service, 1980–1990.
15. Cunningham S: Personal communication. Marriage and Divorce Statistics Division, National Center for Health Statistics, Hyattsville, MD, March 28, 1990.
16. McCleary R, Hay RA: Applied times series analysis for the social sciences. Newbury Park, CA: Sage, 1980.
17. Dixon WJ: BMDP Statistical Software. Berkeley: University of California Press, 1985.
18. Enstad R: Engaged couples still flee state over test. Chicago Tribune April 18, 1988; 2, 7:1.
19. Mercola JM: Premarital HIV screening. (letter) JAMA 1989; 261:2198.
20. Enstad R: AIDS fallout: 1 in 4 leave state to wed. Chicago Tribune July 27, 1988; 1, 1:2.
21. Gostin LO, Curran WJ, Clark ME: The case against compulsory case finding in controlling AIDS—Testing, screening and reporting. Am J Law Med 1987; 12:7–53.
22. Illinois Department of Public Health. Data for first 12 months of mandatory pre-marital HIV testing. Springfield, Illinois, 1989. Personal Communication, April 24, 1990. AIDS Activity Section. Springfield, Illinois.
23. Eisenberg L: The genesis of fear: AIDS and the public's response to science. Law, Med Health Care 1986; 14:243–249.

NOTE

ONLY THE FOLLOWING PAGES OF THIS

PUBLICATION HAVE BEEN FILMED

Cov. 1, Pgs: 3, 4, 11-60